THE physical therapy REVIEW

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Physiology of the Heart and Circulation and Its Clinical Application in Physical Medicine

Part I of a symposium presented at the 27th annual conference of the American Physical Therapy Association in Cleveland, Ohio, June 26-30, 1950

The General Physiology of Heart and Circulation—ALEXANDER

The Influence of Exercise on the Heart—OPDYKE

The Heart as a Factor in a Clinical Exercise Program—BOUMAN

The Physiology and Measurement of Peripheral Circulation —HERTZMAN

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Table of Contents:

Introduction to the Symposium—Harry D. Bouman, M.D., *Coordinator* 451

The General Physiology of Heart and Circulation—R. S. Alexander, A.B., M.A., Ph.D. 452

The Influence of Exercise on the Heart—David F. Opdyke, Ph.D. 462

The Heart as a Factor in a Clinical Exercise Program—Harry D. Bouman, M.D. . . . 468

The Physiology and Measurement of Peripheral Circulation—Alrick B. Hertzman, Ph.D. 471

Contributors to the Symposium 481

Editorials 482

Association Activities 483

Membership News 484

General News 486

Abstracts 487

Index to Current Literature 495

Reprints Available 496B

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Physiology of Heart and Circulation and Its Clinical Application in Physical Medicine

A Symposium

presented at the twenty-seventh annual conference of the

AMERICAN PHYSICAL THERAPY ASSOCIATION

Cleveland, Ohio, June 26-30, 1950

Introduction

Harry D. Bouman, M.D.

Symposium Coordinator

In applying physical modalities to the treatment and diagnosis of patients, our primary interest is often focused on the local effects of the therapeutic or diagnostic procedures used. While these local effects may be the ones desired, one is only too well aware that there are factors involved of a more general nature which can seriously affect and considerably alter the local result.

There is hardly a system that has greater importance as a general regulator system in the application of physical procedures than the circulatory system. While in an exercise program the primary goal is the improvement of muscle function, it is often the circulation that limits our program. Again, in the application of heat, it is the status of the circulatory system which determines, to a considerable extent, the type, extent, and efficiency of our clinical procedure. Also, physical procedures have in many instances proven beneficial in the treatment of diseases of the circulatory system and in the rehabilitation of the patient suffering from a pathological condition of heart and blood vessels. The knowledge of the function of the circulation becomes, therefore, important to the intelligent clinical application of many physical medical procedures.

This symposium is primarily a physiological one. It is often said that what is physiology today, may become clinical medicine tomorrow. This is nowhere more true than in physical medicine. The growth of physical medicine as an intelligent medical specialty may well depend on how solidly its clinical reasoning is based on physiological fact. The careful reader will easily notice that the physiological papers presented in this symposium are based on a large volume of research done in many laboratories. It will also be apparent that many of the physiological facts presented are of immediate clinical importance.

In other cases, however, there is still a gap between physiology and clinical application. To fill that gap more and more research will be needed. The challenge should be apparent to all of us. Every one of us in the field is in a strategic position and can and should contribute to the development of the field. If a symposium like this would be the

stimulus to more and new research it would have proven to be a very real contribution to the development of physical medicine. The stimulating, research-minded questions asked by the audience during these sessions were one of the highlights of the symposium. While it was impossible to include them in the published report, they were an indication to all of us that the future of research in physical medicine looks bright indeed.

This symposium has been the result of the labor and cooperation of many. It has given the opportunity to review a field that is important to physical medicine, to exchange ideas and data, to ask questions, and to receive many stimuli for future research. All of us who attended, as speakers, as discussants, or as members of the audience, owe a sincere vote of thanks to the American Physical Therapy Association and its program committee for the twenty-seventh annual conference for making this possible.

(The C. V. Mosby Company, publishers, allowed us to use the table on page 276 of "Physiology of Exercise" by L. E. Morehouse and A. T. Miller, and allowed us to use Figure 3 from the paper "Applications of Photoelectric Plethysmography in Peripheral Vascular Disease" by A. B. Hertzman and J. B. Dillon, published in the AMERICAN HEART JOURNAL, Volume 20, 1940, page 755.)

The General Physiology of Heart and Circulation

R. S. Alexander, A.B., M.A., Ph.D.

The physiology of the heart and circulation represents a vast subject which cannot be discussed adequately in the brief time at our disposal. Therefore we must confine ourselves to the consideration of only a few of the basic principles involved, realizing that the succeeding speakers will develop more adequately those aspects of the field which are of more direct practical importance to the physical therapist. At the outset I trust we may assume that you already have a general knowledge of the circulatory system, vague though that knowledge may have become in the years since you studied physiology in preparation for your profession. For example, it is not necessary for me to remind you that the circulation of the blood provides for supplying the necessary foodstuffs and oxygen and eliminating waste products and carbon dioxide, all of which are essential for the living processes occurring within the cells of the body. The circulation also plays a uniquely important role in the temperature regulation of the body.

You are also familiar with the basic fact, first appreciated by William Harvey, that the mammalian circulatory system is a closed circuit. More accurately, it is a double closed circuit, comprising the greater, or systemic circuit, which supplies the tissues of the body, and the lesser, or pulmonary circuit, perfusing the blood through the lungs. Throughout the discussion that follows, however, we are going to ignore the pulmonary circulation, and treat the system as though the blood returning to the heart by way of the systemic veins was pumped directly into the systemic arteries. This simplification is often made by physiologists, partly because the pulmonary circulation appears to be extraordinarily competent to take care of itself, so that most practical clinical problems of the circulation are centered on the systemic circulation. Also, it must be confessed that the pulmonary circulation has proved so elusive to experimental analysis that we actually know all too little about it.

Treating the system as a single systemic circuit, then, our discussion must select some starting point in this circuit for consideration. The heart is often regarded as the logical starting point, since it is assumed that the heart is directly responsible for

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pumping the blood through the system, while the arterial blood vessels merely represent the necessary plumbing connections to the pump. Such a concept, however, seriously distorts the true functional relationships in the circulatory system. Actually, the blood supply of our bodies is handled more nearly in the fashion of many city water-supply systems.

For example, here in Cleveland we never have worries about any real water shortage, since Lake Erie is beside us and has never shown any threats of drying up, in spite of the heat of the summer. Pumps are used to pump this water from the lake, but the pumps do not connect directly with the pipes leading into my home or into your hotel room. Rather, the pumps transfer this water into supply reservoirs. The important feature of these supply reservoirs is that they are located at high points in the city, and therefore the Lake Erie water, which was formerly at a low pressure level, has been pumped up to a higher level where it has a proportionately higher pressure. It is to these supply reservoirs that the pipes to our homes are connected, and hence the force directly responsible for providing us with water is the gravitational or "hydrostatic" pressure in the supply reservoir.

Obviously, the function of such a water supply system is basically dependent upon the proper operation of the pumps which raise the water to the supply reservoir, and thus the pumps are, in one sense, the most important unit in the system. Nevertheless, when the hot weather arrives and the consumption of water in the city taxes the system to its maximum capacity, it is not the amount of water in Lake Erie, nor even the operation of the pumps, which concerns us. The major factor we worry about is the water level in the supply reservoir which represents the hydrostatic pressure available for supplying water to our homes. We know that, as long as the supply reservoir contains adequate water, our water supply is assured, but when the level in these reservoirs starts falling to low levels, we are in imminent danger of a serious water shortage.

Transferring this analogy to the circulatory system in our bodies, we find a close parallelism in many ways, even though, as in most analogies, there are some important differences. The job of the heart is not to pump blood directly to the tissues, but rather to keep the supply reservoir well filled with blood under adequate pressure. The supply reservoir in the body is composed of the central aorta and the major arteries. In contrast to the water system, where pressure is maintained by gravity, in the body the pressure in the supply reservoir is maintained by the elasticity of these major arteries. Blood pumped into this central arterial system stretches the elastic walls of these vessels and develops a corresponding tension or pressure which is the driving force responsible for circulating the blood to the tissues.

Viewed in this light, the measurement of arterial blood pressure in patients, as is done so routinely in clinical practice, becomes a direct measurement of the force available to supply the tissues of the body with blood. Indeed, I must re-emphasize the importance of this elastic reservoir function of the major arteries and warn you against thinking of these arteries as mere pipes. In this connection I should point out that in so-called "hardening of the arteries," or arteriosclerosis, this reservoir function of the arteries is deficient, and the arteries actually do become more like pipes. As you well know, the results of this alteration of the system lead to a serious impairment of the proper circulation of blood.

We see, therefore, that, as the heart maintains adequate pressure in the central reservoir arteries, blood is available to supply the vascular beds of the various tissues in the body. The amount of blood flow, therefore, will be determined in part by the arterial pressure. In addition, the flow to any specific organ or tissue will be determined by the size of the opening in the vessel leading to that tissue, together with certain other factors which physiologists refer to collectively as "peripheral resistance." It is unnecessary to go into the biophysical details of this concept of peripheral resistance, since you are all familiar with the common observation that, as one opens a water faucet, the flow of water progressively increases. Here, of course, the pressure in the supply reservoir does not change, but the opening of the faucet is, in fact, decreasing the resistance to water flow from infinity down to lower and lower values. The lower the resistance, the greater the flow of water. In the body, this type of regulation is accomplished by the small arterioles, which have muscular walls and can therefore constrict or dilate. This constriction or dilation alters the resistance, and thereby controls the amount of blood flowing to each organ.

What, in turn, regulates the degree of constriction of these muscular arterioles? Unquestionably, the most important factor governing this action is the degree of activity of the tissue they supply. With cellular activity, there is a release into the surrounding tissue of various metabolites, such as carbon dioxide, organic acids produced by glucose metabolism, organic phosphate compounds, and also potassium. Every one of these metabolites has a characteristic action of dilating the arterioles by relaxation of their smooth muscle. We therefore

have an automatic type of chemical regulation in which the blood supply to a tissue will be constantly regulated to keep pace with its metabolic needs. Since, even at rest, cells maintain a certain basal level of metabolism, a minimal flow of blood is always maintained. The moment cellular activity increases, this local chemical regulation will open up an increased blood supply until the metabolic needs of the tissues are satisfied.

A moment's reflection will reveal that the system, as we have described it so far, has one great limitation. This regulation of flow to individual organs, by adjusting the local arteriolar resistance, will function properly only if there is provision for maintaining an adequate pressure in the arterial supply reservoir at all times. Actually, the volume of blood in this reservoir is definitely limited. If, at any moment, we get wide dilation of a significant number of vascular beds, blood will be drained from the arterial reservoir so rapidly that the pressure will inevitably fall and thus reduce the blood flow. This is prevented normally by having an over-all pressure regulator system under nervous control. There appear to be several of these pressure regulators in different parts of the body, but the most important are a pair of regulators located in the carotid sinuses at the bifurcation of the common carotid arteries into the internal and external carotids. These actually are sense organs which are very sensitive to changes in pressure within the artery. Sensory nerves run from the carotid sinuses to brain centers, which, in turn, relay out over the sympathetic nervous system, where stimulation of the nerve fibers results in constriction of the muscular walls of the arterioles. As pressure falls in the carotid sinuses, this nerve reflex acts to constrict arterioles all over the body, and thereby tends to retain an adequate volume and pressure of blood in the arterial supply reservoir to provide for vital organs. Similarly, a rise in blood pressure tends to inhibit this reflex constriction, to permit dilation of blood vessels and drain off the blood, so as to avoid excessive pressures in the arterial reservoir. In addition to this action on the arterioles, this regulatory system also acts on the heart, by way of the sympathetic and vagus nerves, to speed its rate of beating when pressure falls, and to slow it down as pressure rises. The actual blood flow to a specific organ is therefore determined by the interaction between this over-all nervous regulation of the circulatory system and the local chemical regulation of the supply to the specific tissue.

It should be noted that there are also regulators of the circulation which are governed by the chemical composition of the arterial blood, specifically upon the tension of oxygen and carbon dioxide and upon the acidity of the blood. Time does not permit their detailed consideration except to state that they operate through the nervous system, either by reflex stimulation or by direct stimulation of the brain centers, in a manner quite analogous with the pressure regulators we have just discussed. Other nerve stimuli can influence the circulation, notably stimuli from active muscles which send impulses to the brain centers and thereby contribute to the mobilization of the circulation required for exercise.

In addition, higher brain centers can produce profound effects upon the circulation. A common example of this is the action of emotional excitement in raising blood pressure and speeding the heart rate. A similar cerebral stimulus comes into play with exercise. These responses are obviously for the purpose of meeting the sudden increase in requirements for blood flow which these situations evoke. On the other hand, nerve reactions can produce the opposite effect of a sudden fall in blood pressure due to general dilation of the blood vessels. This type of mechanism explains such things as the fainting reaction to severe pain, and will be discussed in more detail in a few moments.

Our discussion should take us next to a consideration of the capillary bed, which in one sense is the only really important segment of the circulatory system. It is across the thin endothelial lining of the capillaries that the exchange of gases, foodstuffs, and waste products actually takes place, thereby providing for the proper maintenance of the living cells of the body. Obviously, the rest of the circulatory system serves only to assure that this essential process of capillary exchange shall always be maintained. Time does not permit going into the details of this process of capillary exchange. We can only state in passing that the exchange of materials across the capillary endothelial membrane is governed basically by the physicochemical process of diffusion, aided by the interaction of pressure within the capillaries, tending to squeeze fluid into the tissues, and the osmotic properties of the plasma albumin, which acts to retain fluid within the capillaries. The dynamic balance between these forces, plus a number of specialized anatomical arrangements in different types of tissues, admirably provides for bathing the cells of the body with the proper nutrient fluid.

To recapitulate, we must visualize the blood being distributed to the body from the elastic arterial pressure reservoir, the flow to discrete tissues being controlled by the action of the local

muscular arterioles, hence maintaining a perfusion of the capillary beds so as to make possible adequate exchange of materials between the capillary blood and the tissue fluid. Behind all this, of course, remains the pumping action of the heart which provides the driving force to create the arterial pressure head and thereby maintain the circulation. The control of the amount of blood this heart pumps will naturally be the ultimate determinant of the volume of blood perfused through the system. Here, however, we find an important limitation on the heart which often is overlooked. In our earlier analogy with the water supply system, we saw a pumping system drawing from the almost infinite supply of Lake Erie. Such a situation does not hold in the body. The heart does not pump its blood from a vast reservoir, but is strictly limited in its action by the volume of blood returned to it by the venous system. It is obviously impossible for the heart to pump more blood into the arterial system than is returned to it by way of the venous system. At the same time, a normal heart never pumps out any less blood than is returned by the venous system. It is true that under certain circumstances the heart fails to pump out all the blood returned to it, resulting in a pile-up of venous blood. As a consequence, the lungs become congested, leading to severe shortness of breath; the liver becomes distended, and such regions as the ankles become swollen. This, by definition, represents congestive heart failure, and obviously represents a critical derangement of the circulation. So we must always remember that in the normally functioning heart there is pumped into the arterial system no less blood, and no more blood, than is returned by way of the venous system. It becomes evident at once, therefore, that the control of the venous return of blood to the heart is one of the most important aspects of the circulation.

Unfortunately, this is one aspect of the circulation that we still do not understand too well. Perhaps it would be more accurate to say that a good many of us working in the field of circulatory physiology have our own individual understandings of how the system works, but unfortunately our ideas do not agree on all points. Considering the circulatory system as a closed system, the most obvious force pushing blood around through the venous system would be the pressure from the arterial side as originally generated by the heart. The importance of this factor, however, is not as great as might be supposed. The resistance to blood flow through the small arterioles and capillaries dissipates most of the arterial pressure, so that the residue remaining on the venous side is relatively small. Furthermore, although William Harvey was anatomically correct in stating that we have a closed circulatory system, this idea is misleading when applied to the venous system. The venous system not only represents pipes returning blood from the tissue capillaries to the heart, but also contains a number of reservoirs. These function as blood depots, or "storage" reservoirs, and should not be confused with the supply reservoir on the arterial side of the body which we have discussed earlier. Important among these reservoirs are such structures as the skin, the liver, and the gastrointestinal system. For its own metabolism, the skin needs very little blood flow, even though it is richly supplied with blood vessels. Except in its function of temperature regulation, this blood supply of the skin serves no important purpose. When needed, however, this skin circulation may be shut down, its blood dumped into the venous system, and hence returned to the heart. The same type of mechanism appears to function in a number of other organs in the body whose blood supply at rest is significantly greater than their actual metabolic requirements. It should be noted that these storage reservoirs are not necessarily part of the venous system, in the anatomical sense; yet they are conveniently regarded as being part of the venous system in their ability, on the one hand, to discharge their blood volume into the venous return so as to augment the blood available for the heart to pump, or, on the other hand, to store up blood received from the arterial side and thereby cut down on the volume of venous blood being returned to the heart.

Another factor of importance in venous return to the heart is respiration. There is no doubt that respiration tends to aid in venous return to the heart, and, more specifically, that increased respiration tends to augment venous return. Just how this is accomplished is not entirely clear. It has usually been argued that the sucking of air into the lungs in inspiration is accomplished by expanding the chest and thus creating a negative pressure within the chest, and that this same negative sucking pressure must also act to suck blood back toward the heart by way of the venous system. Most of us have been a bit naive in accepting this explanation so glibly. Several investigators have recently argued that the thin-walled veins are collapsible structures, and that you cannot suck blood through collapsible veins any more than you can suck Coca-Cola through a collapsible straw. Certainly this new argument is basically sound, and must be properly evaluated and tested before we can be sure how important this sucking mechanism is, if indeed it functions at all. A less debatable mechanism by

which respiration increases venous return is through the action of the diaphragm. As the diaphragm descends with inspiration, it squeezes blood from the abdominal viscera, especially the liver and intestines, and thereby increases venous return to the heart.

Whatever the whole story may be as to the actual mechanics involved, the fact that increased respiration does increase venous return is certainly an important factor to be remembered, especially when one bears in mind that typically when there is need for increased circulation of blood, as, for example, in exercise, we do observe a marked increase in respiration.

An additional factor of importance in aiding venous return, which is also of importance in exercise, is the massaging or "milking" action of contracting muscles. When muscle fibers contract, they bulge, and must thereby compress the veins in that vicinity. This must act to squeeze blood out of these veins locally and, at least in the case of veins which have valves, must act to propel blood back toward the heart. This mechanism obviously would be of most importance in the limbs, although it also appears to play a role in such structures as the intestines. Also, it applies only to veins deep within the muscle tissue, and not to the superficial veins, the latter being attested to by the frequent failure of the superficial veins of the legs to properly return blood to the heart, as is often observed clinically. In addition, it should be emphasized that this mechanism works only with active muscular movements, as in walking or running. It has been rather clearly demonstrated that milking of venous blood is not accomplished by steady, maintained, tonic contraction of muscle, as in standing erect, or in maintained muscle tension against resistance as frequently employed in physical therapy. The importance of this latter fact will be brought out by Dr. Opdyke later this morning.

Most of what we have said about the venous system applies also to the lymphatic system. Indeed, from a physiological standpoint the lymphatic system should be regarded as a subdivision of the venous system, since it picks up its fluid from the tissue spaces surrounding the capillary beds, as the venules do, and returns this fluid to the great veins leading to the heart.

Now, consider the heart itself, which obviously is the pump constituting the basic driving force behind this entire system. From what has been said already, we have a pretty good idea of the job which the heart is called upon to perform. It must be capable of an extraordinarily flexible activity to continually adjust its pumping action to accommodate the venous return from moment to moment, and pump this blood into the arterial supply reservoir at whatever pressure happens to be in the supply reservoir. It is not surprising to find that the mechanism for this exquisite control of the pumping action of the heart is self-contained within the heart itself, and is, therefore, entirely automatic. This autonomy of the heart in regulating its pumping action cannot be overemphasized, specifically in relation to its independence from its nerve supply. True, the nerves to the heart can influence its rate of pumping, and appear to have some minor influence on the force of its contraction. The basic mechanism determining the force of contraction of the heart, however, has nothing to do with its innervation.

This basic mechanism controlling heart action is extremely simple and, to the physical therapist, exceedingly commonplace. You are all familiar with the fact that it is not possible to obtain much work from a muscle unless that muscle contracts against a significant load. Within limits, the greater the load presented to a muscle, the greater the force it exerts with contraction. This applies to skeletal muscle; it applies to smooth muscle; and it applies to cardiac muscle. This principle accounts for the justified emphasis on resistive exercise for muscle development which you are now stressing in physical therapy. It is also the guiding principle determining the action of our hearts. More specifically, we know that the degree of stretch placed upon the muscle fibers, at the moment they start to contract, determines the force which they will exert with contraction. Although this is one of the earliest recognized principles in physiology, the full importance of this principle in determining heart action was first appreciated by Starling, the great English physiologist of the first quarter of this century, and hence it is frequently spoken of as "Starling's Law of the Heart."

The actual application of this principle to the pumping action of the heart is very easy to visualize, provided we forget any notions we might have acquired to the effect that the auricles pump the blood into the ventricles. This anatomical inference has virtually no basis in fact; in man the auricles do not effectively pump anything anywhere. Indeed, the mammalian auricle is of so little functional importance that we will not even take time to discuss its function here. For the moment, let us forget about the auricles, and visualize the great veins emptying directly into the ventricle of the heart. During the period between beats, when the heart is relaxed (diastole), the heart will be filled with blood returning by way of the venous

system. The greater the rate of return of venous blood, the more the heart will be filled. In other words, the greater the venous return, the more the heart fibers will be stretched during diastole. Now, at the moment the heart starts its contraction (systole), the force exerted during systole will be determined by the degree of stretch of the muscle fibers produced by venous filling. Thus we see that the pumping action of the heart is directly regulated by the venous return. With a low venous return, the pump exerts little force; with increased venous return, the action of the pump is automatically increased by this stretch principle so as to keep pumping into the arterial system the volume of blood delivered to it by way of the venous system.

Compensation to increased arterial pressure is accomplished in much the same way. It is now generally recognized that the heart does not empty itself completely at each beat, but there is left within the heart chamber a certain residual volume of blood. A major factor in determining this residual volume is the existing arterial pressure. The higher the arterial pressure, the greater the residual volume. Now it follows that this residual volume will add to the venous return, to stretch the heart during diastole. Thus the greater the arterial pressure, the greater the residual volume; the greater the residual volume, the more the heart will be stretched during diastole with a constant venous return; and the greater the stretch on the heart muscle, the greater will be the force exerted in contraction to work against the arterial pressure. In addition to this "loading" principle, there is also evidence that heart muscle, like skeletal muscle, responds to an "after load." In other words, not only is the heart contraction more forceful when presented with an initially increased load, but it will also contract more forcibly when confronted with the increased tension required by an elevated arterial pressure. It is by these intrinsic physiological mechanisms that the normal heart is nicely regulated to carry out the job of pumping the required volume of blood through the system.

We should note, in passing, that this principle of cardiac compensation has certain limitations as with any compensatory mechanism. You know that, with skeletal muscle, there is a limit in loading a muscle beyond which the muscle becomes overstrained and is incapable of developing adequate force. You also know that the danger of overstraining a well-developed muscle is not too great, while it is rather easy to overstrain a weak muscle. The same applies to the heart. A normal, healthy heart has a tremendous reserve of compensation available for handling an increased load. Not so with the diseased heart. Its reserve is cut down to the point where it is capable of very little compensation. Any appreciable increase in the load placed upon a diseased heart may result in overstretching the muscle beyond its compensation range; thus the force of the heart beat drops off, in spite of increased venous return. Exactly this mechanism is involved in the so-called "decompensation," or acute congestive heart failure, which anyone with a seriously diseased heart may encounter when he makes the mistake of engaging in strenuous physical exertion.

So far we have said little about the rate of the heart beat. Actually, there is a tendency to overemphasize the importance of heart rate changes in circulatory dynamics. As with other factors, changes in heart rate cannot significantly increase the amount of blood pumped by the heart, unless venous return also changes. If venous return remains constant, an increase in heart rate will only result in less blood being pumped per beat, the total cardiac output remaining unchanged. Similarly, if venous return is adequate, a slow heart can pump very large amounts of blood by the compensation principle we have just discussed. Evidence of this relationship is seen frequently in cardiac patients who have disturbances in heart rate. Many ambulatory patients may go about their business with heart rates remaining between 150 to 180 per minute, while others make out reasonably well with heart rates around 55 per minute. Both types of patients may have adequate peripheral circulation of blood. These patients are of concern to the cardiologist, not because of the abnormal heart rate itself, but rather because the disturbed heart rate is a sign of a diseased heart and other consequences of this disease process are more to be feared.

There are two ways, however, in which changes in heart rate influence the efficiency of the circulation. First, heart rate alters the efficiency of the heart itself. The normal resting pulse rate of around 70 per minute does not represent the optimum rate in terms of mechanical efficiency of the heart muscle. This efficiency is increased by accelerating the rate of the heart up to about 120 per minute. Just where the optimum rate lies has been debated, and certainly varies with different individuals. There is no doubt, however, that at rates above 150 per minute the mechanical efficiency of the heart seriously deteriorates. This is the reason why cardiologists are anxious to slow the heart of a patient whose pulse has risen to very high rates. The low mechanical efficiency of these high heart

rates, coupled with an already weakened heart muscle, may precipitate acute heart failure.

In addition, moderate increase in heart rate allows the heart to pump out large volumes of blood at lower levels of venous pressure. The mechanism involved here will become clear if one works out the influence of heart rate on the Starling law principle. A slow heart can pump large volumes of blood only by maintaining a high venous pressure to provide adequate stretch of the heart muscle during its filling phase. This elevated venous pressure can be obtained only by damming back a certain amount of the circulating blood volume on the venous side. By accelerating the heart rate, the heart can pump the same amount of blood, without requiring as high a venous pressure to distend it during diastole. As a result, the circulation of the blood is more rapid, and distribution to the periphery occurs more efficiently, without blood being logged up in the venous system. For these reasons, we find that heart rate is altered by means of nerve impulses over the vagus or sympathetic nerves to aid in adjusting the circulation to the demands of any particular moment.

Now, to put all this together. To attempt to understand the circulatory system by examining the function of its individual components is not to understand it at all. Until we can put the system together into a functioning unit, we have no true concept of how the system really works. To aid in this synthesis, diagrammatic sketches have been given. I should emphasize that these diagrams are ridiculously oversimplified, and, in themselves, can be very misleading unless one pays careful attention to the principles they are designed to demonstrate. In Figure 1 we have pictured the normal resting circulation. The heart is represented above in its contracting and relaxing phases. Blood is pumped clockwise into the elastic arterial system, as indicated by the arrow. It is then distributed to the capillary beds by the arterioles which run horizontally from the main arterial trunk to the individual beds. The resting supply to each capillary bed will be regulated by arteriolar constriction, depending upon the needs of that tissue under basal conditions. In the diagram, the lower capillary bed has been pictured as requiring less blood than the upper capillary bed. Note also the blood reservoir. This does not represent any discrete organ in the body, but is merely symbolic of all those vascular beds of the body which, at rest, tend to pool a volume of blood which is thereby taken out of the active circulation.

Next let us consider what happens in exercise, as shown in Figure 2. Actually the whole story of

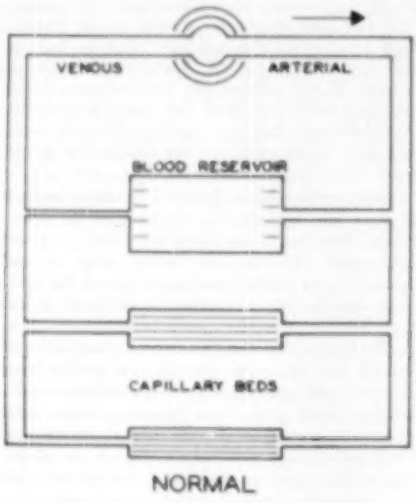

Figure 1.

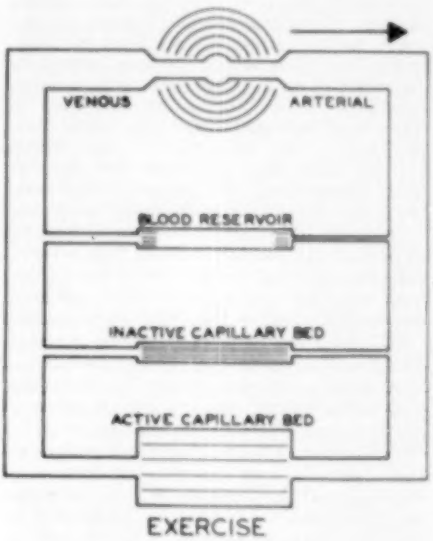

Figure 2.

what happens to the circulation in exercise is extremely involved, as will become evident when Dr. Opdyke discusses this aspect in somewhat greater detail. Only a few points warrant emphasis at the moment. Comparing Figure 2 with Figure 1, it is evident at once that there has been a tremendous change in the activity of the heart. The heart is being distended much more, and contracting far more forcibly. This is primarily the result of emptying of the blood storage reservoirs into the venous return, as shown. Since venous return is so very much greater, by Starling's law, the force of the heart, and hence the volume it pumps, must be greatly increased. It will also be noted that the heart, in exercise, contracts to a smaller residual volume, in spite of the increased load. This appears to result from the stimulating action of the heart muscle, derived from the sympathetic nervous system, as well as some adrenalin which is released into the blood stream by the adrenal gland. As a consequence of the greatly increased volume of blood being pumped out by the heart, arterial pressure tends to rise, and arterial blood flow increases tremendously. The arterioles leading into the inactive capillary beds and the reservoir organs tend to shut down, so as to divert blood to where it is needed. In the active capillary beds, however, local accumulation of tissue metabolites results in a tremendous dilation of the arterioles supplying the capillary bed of the active tissue, which in this case would be represented by the skeletal muscle. As a result, the tremendous cardiac output is largely perfused through the active tissue. This blood pours out of the active capillary bed into the venous system, and, aided by increased respiration and the massaging action of the contracting muscles, flows back in large volumes to the heart. Hence the cycle is maintained as long as the exercise continues.

Now turn to consider the circulation in the hypertensive patient at rest, shown in Figure 3. Chronic high blood pressure, or "hypertension," actually embraces a number of different forms of vascular disease, the details of which do not concern us at the moment. For the purposes of simplification, we therefore picture hypertension as a purely arteriolar disease, as represented by the thickened arterioles. Because of the small bore of these arterioles, and hence their large resistance to blood flow, blood tends to back up in the arterial supply reservoir and place it under high pressure. For reasons that are not entirely understood, the physiological mechanisms, which usually act to prevent such a rise in arterial pressure, do not function at normal levels in the hypertensive patient. We thus have a persistently high arterial pressure, with consequent changes in the heart. When forced to contract against this high pressure, the heart does not empty as much as normally, and therefore the residual volume increases. This residual volume, plus the normal venous return, stretches the heart during diastole, so that at all times the heart tends to be much more dilated than normally. It is this physiological dilation, plus some anatomical hypertrophy, which explains the large heart size observed by X ray or physical examination of the hypertensive patient. The dilation, by placing a greater stretch on the muscle fibers, acts to increase the force of the heart beat, so that it can contract effectively against the high arterial pressure and maintain a reasonably normal circulation of blood. Note that the blood reservoirs, the capillary circulation, and the venous circulation is pictured as being normal in the hypertensive patient. This appears to be essentially true in the patients we see clinically, until the disease becomes very far advanced. We see, therefore, that by cardiac compensation the heart of the hypertensive patient can maintain a reasonably normal circulation of blood, in spite of the persistent resistance it must work against.

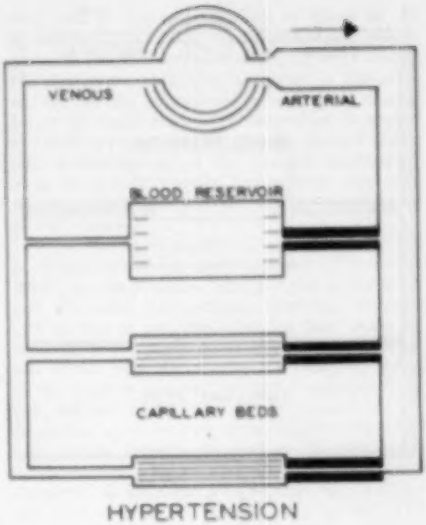

Figure 3.

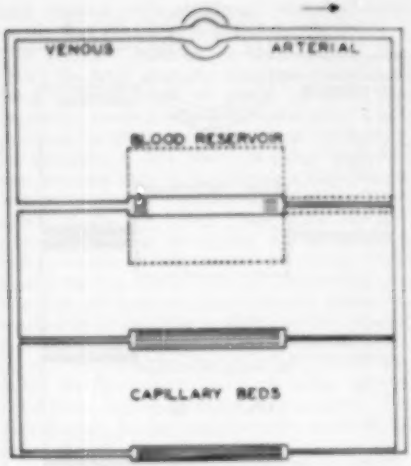

Figure 4.

But now compare Figure 3 with Figure 2. Here we see the real problem of the hypertensive. What will happen with exercise? The compensatory ability of the hypertensive's heart has already been taxed by the high peripheral resistance at rest. The added venous return of exercise may well overload the heart. In addition, granting that the heart is able to demonstrate some additional reserve and pump this additional blood into the arterial side, what will happen to the already excessive arterial blood pressure when this greater cardiac output is pumped into the supply reservoir? We trust that a little study of these diagrams will make quite clear why the hypertensive in the earlier stages exhibits a reduced exercise tolerance, and why, as the disease progresses, he becomes more and more vulnerable to the imminent danger of strenuous exercise precipitating one of two eventually inevitable consequences: either overloading of the heart so as to produce decompensation and acute congestive heart failure, or a rise in arterial pressure to the point where the system bursts in its most vulnerable spot, which is usually in the cerebral vessels.

Incidentally, the problem of the heart which we have described for the hypertensive is basically the same for any form of heart disease. Almost all forms of heart disease involve, at least in their later stages, a dilation of the heart in attempt to compensate for the load placed upon it by the weakening of its functional capacities. Consequently, the strain placed upon such hearts by strenuous exercise results in the same essential problem as confronts the heart of the hypertensive. Without adequate cardiac reserve, the normal circulatory compensation to exercise is not possible.

Finally, we see the converse of this relationship in Figure 4, which portrays the circulation in hypotension. Hypotension, or a seriously lowered blood pressure, may result, in general, from one of two causes. First we might consider any factor leading to a serious loss of blood from the circulatory system. This obviously results from hemorrhage, and may also be observed in patients with severe burns, or patients who have lost large amounts of fluid from the body by persistent vomiting, severe diarrhea, or even profuse perspiration, which may accompany heat prostration or high, spiking fevers. Their venous return is low because of inadequate blood volume. The blood reservoirs in these conditions, indicated by the solid lines in Figure 4, have already emptied themselves into the venous system in order to compensate for the loss in blood volume. Since venous return is low, it follows from Starling's law that heart action will be weak and arterial pressure and flow reduced. Since arterial pressure and flow are drastically lowered, there obviously can be little flow to the capillary beds. The circulation may be able to limp along in this condition for a brief period, but it is obvious that if the metabolic needs of the tissues are not provided for, life cannot continue. We thus have the development of so-called "irreversible shock," which can be prevented only by rapid diagnosis of such conditions and immediate transfusion, or similar measures, to restore an adequate circulatory volume.

Presumably this extreme form of shock is not often encountered by the physical therapist. Nevertheless, it must be remembered that any patient convalescing from serious illness or major surgery will tend to have a blood volume that is below normal. This is compensated by shutting down the storage reservoirs so as to maintain adequate venous return, adequate arterial pressure, and an adequate peripheral circulation. Without ample reserve of blood in the storage reservoirs, however, one must anticipate some impairment in the exercise tolerance of such patients.

Another form of acute hypotension is represented by the dotted lines in Figure 4. For some reason, various organs of the body suddenly become storage reservoirs, and pool so much blood

that there is no longer adequate blood left in the active circulation. This condition is sometimes referred to as "intravascular hemorrhage," since, from the standpoint of the immediate effects on the active circulation, its consequences are exactly the same as with actual arterial hemorrhage. This situation may readily be precipitated by nerve reflexes, acting to open up the reservoir organs, and is responsible for the fall in blood pressure and fainting reaction which may be associated with severe pain or emotional disturbances. In these instances the reaction is only a transient one, and is rarely of serious consequence, in itself. At other times, this mechanism may arise with poisoning, as from the toxins liberated in massive bacterial infections, in which case the situation may be very grave.

A special example of this latter type of hypotension, with which you are particularly familiar, is postural hypotension. It appears that the various vascular reflexes, responsible for maintaining proper tone of the arteriolar musculature, demand repeated use in order to remain in proper coordination. Just as a brief period of disuse of skeletal muscle destroys precise neuromuscular coordination, so the coordination of the sympathethic nervous system seems to disappear after a brief period of disuse. Yet such a coordination is essential, if the body is to make the important adjustments in vascular tone to compensate for the gravitational action on blood flow as a result of changes in posture. It is for this reason that most patients who have been on a period of strict bed-rest of any duration have difficulties in postural adjustments. The dizziness and faintness which such patients experience when they arise is due to a pooling of blood in the lower parts of the body. As a consequence, venous return to the heart drops sharply; the heart output and arterial pressure drop as shown in Figure 4, and the fainting is produced by inadequate circulation to the brain. Medically, this problem is usually of trivial consequence, but, to the physical therapist, re-coordination of the vascular innervation, so as to alleviate postural hypotension, may well represent the major problem in the rehabilitation of such patients.

With these diagrammatic representations of some of the more important problems of the circulation, we complete our brief glimpse of the heart and circulation. In conclusion, however, we must confess that in our discussion we have made no mention at all of the most important circulatory bed in the body. Obviously, the pumping action of the heart is essential for this circulatory function, and yet the function of this pump is in turn dependent upon its own blood supply—the coronary system. No vascular bed in the body must be maintained with an adequate flow of blood as constantly as must the coronary system supplying the heart muscle. No vascular bed in the body makes such efficient use of the blood supplied to it as does the coronary system. As a consequence, no vascular bed in the body demands more exacting regulation of an adequate supply, moment by moment, as does the coronary system. But then, this is another story. As the succeeding speaker is a recognized authority in this field, it would be out of place for me to tell you the story of the coronary system. I know he will develop that topic much more adequately than could I.

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The Influence of Exercise on the Heart

David F. Opdyke, Ph.D.

The study of physiologic mechanisms, which operate in response to the need of the individual, is indeed a fascinating one. A bout of exercise not only calls into play innumerable adjustments, but, in addition, results in a coordination of various functions that is without biologic parallel. We are aware that the convalescent patient cannot exercise to the same degree as the healthy individual. We also note that some healthy people can perform greater feats of exercise than others can, which accounts for champions in sports. After making due allowance for mechanical factors, the *exercise tolerances* of the convalescent patient and the healthy person are limited by the same factors, differing only in degree.

The factors limiting the exercise tolerance reside in the circulatory system. However, before one can appreciate how the limitation of the exercise tolerance comes about, one must be acquainted with some of the physiologic events associated with the act of exercising. Table I has been constructed especially for this purpose. It shows many, but not all, of the adjustments that take place during a bout of exercise. The lower half of Table I is of immediate interest to us.

The characteristic of exercise is obviously the use (contraction) of skeletal muscle. We may reason that physiologic mechanisms will operate with the objective of making possible the maximum use of the muscles for the longest possible time. Referring to Table I, we see that the first chemical reaction in the process of muscular contraction is an anaerobic breakdown of the substance, adenosinetriphosphate, which loses a phosphate group and gives up energy. The second reaction, the conversion of phosphocreatine to creatine, free phosphate radical, and energy, is likewise carried out in the absence of oxygen. These reactions provide the primary source of energy for muscular contraction.

The substances adenosinetriphosphate and phosphocreatine are, so to speak, the real fuel of muscular contraction. However, the supply of these fuels in the muscle cell is limited, and a very short period of contraction will use up these compounds, unless chemical reactions are going on at the same time which replenish the supply. The reactions which result in the formation of more adenosinetriphosphate and phosphocreatine are oxidative in character. The chief substance oxidized is glucose, although the oxidation is not direct, many intermediate steps being necessary before glucose is finally broken down into carbon dioxide, water and energy.

The purpose of the whole oxidative cycle is to provide the energy to cause the re-formation of the anaerobically-acting fuels, adenosinetriphosphate and phosphocreatine. An adequate and continuous supply of oxygen to the exercising muscle is indispensable, if exercise is to be maintained for any length of time. Whether or not the supply of oxygen is adequate depends upon the circulatory system to a great degree, assuming the pulmonary system to be functioning normally. Since the O_2 is carried to the tissues in loose combination, with the hemoglobin contained in the red blood cells, the volume of blood, per unit of time, delivered to an exercising muscle system will determine the amount of oxygen made available to that system during that time.

Referring again to the schema showing many of the adjustments made during a bout of exercise, we see that many of these adjustments have to do with providing a greater volume of blood flow where it is needed. Note particularly that the heart pumps more blood (a result accomplished by increasing the venous return by the massaging action of muscular contraction and the emptying of blood reservoirs, such as the skin circulatory bed), increasing the heart rate by reflex action and by increasing the volume of blood expelled by the heart at each beat. Not only is more blood pumped by the heart, but the blood is rerouted so that a greater proportion of the total flow is diverted to the exercising muscles. First of all, the vessels, in regions of the body which are not actively participating in the exercise, receive a lesser proportion of the total amount of blood pumped—the abdominal viscera, for example. This condition may be the result of a constriction of arteriolar

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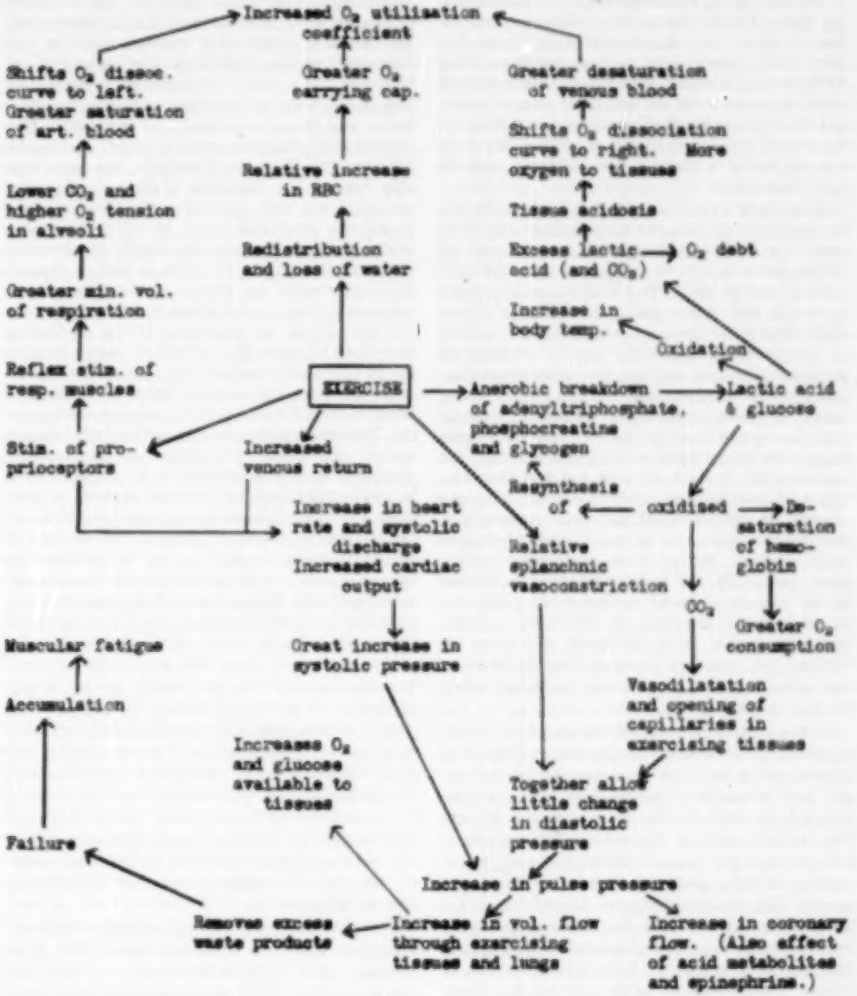
TABLE I

Sequence of Physiological Events During Acute, Moderate or Severe Exercise


Increased O_2 utilization coefficient
Shifts O_2 dissoc. curve to left. Greater saturation of art. blood
Greater O_2 carrying cap.
Greater desaturation of venous blood
Shifts O_2 dissociation curve to right. More oxygen to tissues
Lower CO_2 and higher O_2 tension in alveoli
Relative increase in RBC
Tissue acidosis
Greater min. vol. of respiration
Redistribution and loss of water
Excess lactic acid (and CO_2)
O_2 debt
Increase in body temp.
Oxidation
Reflex stim. of resp. muscles
EXERCISE
Anaerobic breakdown of adenyltriphosphate, phosphocreatine and glycogen
Lactic acid & glucose
Stim. of proprioceptors
Increased venous return
Resynthesis of
oxidized
De-saturation of hemoglobin
Increase in heart rate and systolic discharge
Increased cardiac output
Relative splanchnic vasoconstriction
CO_2
Greater O_2 consumption
Muscular fatigue
Great increase in systolic pressure
Vasodilatation and opening of capillaries in exercising tissues
Accumulation
Increases O_2 and glucose available to tissues
Together allow little change in diastolic pressure
Failure
Increase in pulse pressure
Removes excess waste products
Increase in vol. flow through exercising tissues and lungs
Increase in coronary flow. (Also effect of acid metabolites and epinephrine.)

vessels in such areas, but it is likely that this is more apparent than real. The effect of metabolites, chiefly carbon dioxide, adenylic acid, the three carbon acids of the lactic-acid type, the increase in temperature, and possibly other factors, results in a dilatation of the blood vessels in the exercising tissue. In this manner the resistance to the inflow of blood is reduced, and more blood will enter these regions. Also, such a process probably accounts for the diversion of blood from nonactive areas. As a result of the increased cardiac output and the decreased inflow resistance, an increase of the arterial pulse pressure occurs which is obviously a potent factor in pushing more blood through the exercising tissue.

As a result of all these factors, the blood flow through exercising muscle may increase tenfold or more. Yet, even such a tremendous flow may not be enough to supply the oxygen need of the contracting muscle fibers. This inadequacy is pointed up by the fact that a person can go into oxygen debt—that is, he can use up the anaerobic sources of energy faster than they can be restored by oxidative processes, and, his O_2 consumption continues to be increased for a time after exercise ceases. A person engaging in exhaustive exercise may run as much as 14 to 15 liters of oxygen behind his needs for that particular exercise. All of this points to the hard, cold fact that the duration and intensity of exercise is limited by the amount of oxygen available, and, furthermore, that the limiting factor in the amount of oxygen made available resides in the circulatory system. More specifically, the exercise tolerance is limited by the amount of blood the heart can pump, or, to put it in another way, by the cardiac output. Recognizing this, it is, therefore, imperative to consider the heart as a pump and to inquire as to how it works and why it becomes inadequate when exercise exceeds certain limits.

It is customary to speak of the amount of blood pumped by the heart as the cardiac output. This is expressed in terms of liters pumped per minute and may be calculated by multiplying the volume pumped per beat by the heart rate per minute. The cardiac output of the average adult at rest is 5 to 6 liters per minute, although during heavy exercise it may amount to 25 to 40 liters per minute. The stroke volume, in simple terms, depends upon two factors: the amount of blood returned to the heart by the systemic venous system (for the heart cannot pump more blood than it receives), and the ability of the heart to eject blood from its cavities against the pressure of the blood contained in the great arteries.

The contraction of the heart muscle (myocardium) on the blood contained in its cavities develops pressure. It is the higher pressure in the ventricles, compared with that in the great arteries, that causes the blood to be ejected from the heart. The force of contraction depends upon a number of factors: the number of muscle fibers contracting, the extent to which they contract, and the synchronicity of contraction of the various fibers. However, the number of fibers contracting at each beat of the heart is relatively constant (all-or-none law), and the synchronicity of individual fiber contractions does not vary a great deal under different circumstances. Therefore, the most variable factor is the extent to which each fiber contracts. As has been pointed out in the preceding paper, the force and extent to which each fiber contracts depends upon the length of the fiber just prior to contraction, which, in turn, is dependent chiefly upon the degree of dilatation of the ventricle. Going a step further, we can also say that the degree of shortening of the myocardial fiber depends upon the amount of energy released by the chemical reactions of contraction. As in skeletal muscle, the chemical reactions responsible for providing the energy of contraction are anaerobic; however, cardiac muscle differs from skeletal muscle. Apparently, the supply of adenosinetriphosphate and phosphocreatine in cardiac muscle is much more limited than in skeletal muscle, or its rate of use is much greater. As a consequence, cardiac muscle cannot operate in the absence of adequate oxygen as long as can skeletal muscle. To put it another way, cardiac muscle cannot incur an oxygen debt. This makes it imperative that some provision be made for increasing the supply of oxygen to the heart muscle, to an extent commensurate with the greater demands made upon it. The heart muscle does not benefit by the oxygen contained in the blood within its cavities, but rather the myocardium is supplied by the coronary blood vessels distributed through the walls of the heart. Therefore, as in the case of skeletal muscle, the amount of work that the heart can do is limited by the amount of blood which can be delivered to it through the coronary system. The adequacy of the coronary system, therefore, in the final analysis, becomes the limiting factor in determining exercise tolerance.

The main coronary arteries originate from the aorta, just above the semilunar valves. The right coronary artery supplies the right ventricle, and also the posterior half of the interventricular septum, while the left coronary artery supplies the left ventricle and the anterior half of the septum.

Each of these arteries, after subdividing a few times, courses directly into the muscle. The arterial twigs may pass through one or more muscle layers before dividing again. Finally, the arterioles break up into a capillary network whose vessels run parallel to the direction of the myocardial fibers. The coronary blood is returned to the right atrium by way of the coronary sinus, which drains chiefly the left ventricle, the anterior cardiac veins, and the Thebesian vessels.

The blood flow through all of the coronary system has been estimated at 5 to 10 percent of the total cardiac output at rest. How much coronary flow is increased during exercise we do not know, although it is certain that flow is increased. The increase in flow results from the interaction of three factors: (a) the pressure head in the root of the aorta, (b) the cross-sectional diameter of the coronary vessels, and (c) the effect of extravascular support of the coronary vessels. When the mean aortic pressure is increased, as it is during exercise, more blood is pushed through the coronary system. Of all the factors increasing the rate of flow, this is perhaps the most important one. The second factor is the diameter of the coronary arterioles, and, perhaps, the capillaries. The greater the total cross-sectional diameter of these vessels, the lower the resistance to the onward flow of blood. Thus, if the vessels dilate, and they are thought to do so during exercise, all other things being equal, the amount of blood flowing through the coronary system would be increased. The third factor is one that is somewhat difficult to assess. As the ventricular muscle contracts, a compressive force is exerted on the vessels running in the walls of the ventricles. This compression acts to squeeze down on the vessels and to prevent blood from entering from the arterial end; but, at the same time, it tends to squeeze blood toward the venous end. During exercise, the number of compressions per minute is increased, due to the increased heart rate, and each compression exerts greater pressure, due to the greater force of contraction. Our best experimental evidence to date indicates that this action does not increase coronary flow, but rather hinders it.

Now, let us examine in detail the relation between exercise and the rate of coronary flow. As the result of exercise, the amount of work that is done by the heart muscle is increased. Work can be defined as the displacement of a mass as a result of the application of a force. In the case of the heart, the displaced mass is the blood pumped by the ventricles, and the force is the pressure exerted on the blood within the ventricular cavities by the contraction of the heart muscle. For comparative purposes, we can roughly approximate the work done by the heart by multiplying the volume of blood pumped per minute by the mean arterial pressure. As we have indicated before, the amount of blood pumped during exercise is greatly increased over that pumped at rest. The increase of cardiac output comes about by increasing both the amount of blood ejected at each stroke of the heart, and the number of strokes per minute. Mean arterial pressure, the pressure against which the heart must work, also increases during exercise, due chiefly to the increase in stroke volume and increased heart rate. During rest, the heart may perform work at the rate of 6 kg. meter per minute, but, during exercise, the work output may increase tenfold or more. As in skeletal muscle and other mechanical machines, the work output of the heart is not accomplished with 100 percent efficiency; only a small fraction of the total energy-expenditure appears as work. The resting heart has a very low efficiency rating, somewhere around 3 percent; however, during exercise, the efficiency increases to a respectable 20 percent or more.

Coronary flow is also increased during exercise. In the first place, arterial pressure is increased, thus increasing the pressure head, which pushes blood into the coronary system. Recent evidence has been adduced which leads to the conclusion that the resistance to flow through the coronary vessels is decreased. This decrease in flow resistance is presumably due to a widening of the coronary vessel bed. There are two mechanisms which could be responsible for this: first, a relaxation of the vasoconstrictor tone of the coronary vessel walls, or an active dilatation of the vessel. These effects would be brought about by reflex action as the result of nerve impulses arising from the action of skeletal muscles. The second possibility is that the vessels are dilated as a result of the action of metabolites, hormones, or relative lack of oxygen. It is known that a number of substances liberated by contracting muscle will act to dilate coronary vessels—lactic acid, for example. The hormone, epinephrine, which is liberated from the adrenal medulla during exercise, is reputed to be a coronary dilator. Anoxia, the lack of adequate oxygen, is a very potent dilator of coronary vessels. Some work indicates that the rate of coronary flow is adjusted by the relative rate at which oxygen is needed, indicating that a condition of relative oxygen-lack is a potent force in increasing coronary flow. Since both the heart rate and the extravascular compression-effect on the coronary vessels are increased, one would expect a decreased coronary flow as the effect of these factors. However,

the effect of increased pressure head, and the decreased flow resistance due to dilatation of the vessels, seems to greatly outweigh this effect.

In summary, then, the effect of exercise on the heart is to increase the work load on the heart, by virtue of increasing its output and the pressure against which it works. Concomitant with the increase in work, the coronary flow is also increased. The increase in coronary flow is a necessary event if the heart is to maintain an increased work output for any period of time, since an adequate oxygen supply depends upon an adequate coronary flow.

The fact that the work output of the heart increases with exercise indicates that a greater amount of energy must be expended by the heart muscle. Since the energy is ultimately derived from oxidative reactions, it follows that the oxygen requirement of the heart is also increased. Because the oxygen must be transported by the coronary circulation, the question is raised as to whether or not the coronary circulation is adequate at all times to furnish the required additional oxygen. Just what the relation is, between the amount of work done by the heart and its oxygen requirement, is not known with certainty; however, it is known that the heart muscle extracts a large amount of oxygen from each unit of blood passing through it, even during rest. The average oxygen saturation of coronary sinus blood is about 23 percent, whereas mixed venous blood is usually about 70 percent saturated. This indicates that if the heart muscle needs more oxygen it must obtain it by increasing the coronary flow, since very little more can be obtained by extraction from the resting blood flow. It is also true that, as the contractile force of the failing muscle decreases, the oxygen requirement continues to increase, indicating a necessity for an even greater coronary flow. As in the case of skeletal muscle, it is probable that the requirement of the myocardium for oxygen increases out of proportion to the amount of work done. Furthermore, it is probable that every individual has a critical limit to which his coronary flow can be increased, thus limiting the work capacity of his heart, and, thus, his exercise tolerance. This limit applies to the individual with a normal, healthy heart, as well as to the individual with heart disease; it is merely a matter of degree.

Unfortunately, in the case of severe exercise, the increase in coronary flow apparently is not sufficient to supply all the oxygen needed by the heart, even in the case of a normal individual. Regardless of the physical state of the individual, some severity of exercise can be reached at which this situation will obtain. It is also unfortunate that no physiological means of increasing the rate of coronary flow, over and above what adjustments the heart itself can make, are known. We are, therefore, faced with the situation of making the best use of the coronary flow existing under any particular conditions.

It is always desirable that any bodily mechanism perform efficiently and possess some reserve in order to meet occasional, out-of-the-ordinary demands that may be placed upon it. The heart is not an exception to this need, in fact, it is the one organ of the body which must necessarily possess these attributes. Therefore, it has always been of interest to know whether or not, in some way, the work capacity of the heart could be improved, and its reserve increased, by a planned program of physical exercise. In the case of the normal, healthy heart the answer is "Yes."

Let us examine the effects of a program of training on a normal heart. Suppose we subject an individual of sedentary habits to a training program for a period of six months. The daily bout of exercise might be running on a treadmill at known speed. Comparing his performance before and after the training period a number of improvements would be noted. For convenience, these have been listed in Table II. We are chiefly interested in items 6, 9, 10, and 11, since these pertain more particularly to the heart.

Because oxygen consumption and cardiac output run parallel, roughly, during heavy exercise, the increase of maximal oxygen consumption (6) indicates that the subject has increased his maximal cardiac output, and, hence, his exercise tolerance. Since there is no significant difference between mean blood pressure of trained and untrained subjects when doing exhausting work, and if we recall our simple approximation of cardiac work (C.O. x MBP), we see at once that the heart of the trained individual is capable of doing much more work than that of the untrained person, because cardiac output is increased, and mean blood pressure is unchanged. The primary effect of training on the heart, then, is to increase its work capacity. It might be said, in passing, that running exercises are best for bringing about the changes noted in Table I. Weight-lifting exercises, for example, do not result in such a development.

We may inquire what adjustments the heart makes in order to acquire the capacity for additional work. As I have pointed out previously, there is no evidence that the absolute rate of cor-

TABLE II—TRAINING[1]

Summary of the Effects of Training on the Physiological Systems. The effects of frequent and regular exercise on the physiological systems of the body have been observed repeatedly during standard work experiments. Among the changes which have been reported are the following:

1. slight increase in body weight
2. longer duration of effort before exhaustion
3. slight decline in the rate and depth of breathing at rest
4. decreased pulmonary ventilation during moderate work
5. ability to attain a greater minute-volume of ventilation in exhausting work
6. ability to attain a greater oxygen consumption during exhausting work
7. greater mechanical efficiency, as indicated by a lower oxygen consumption for a given amount of work
8. greater utilization of anaerobic energy reserves
9. lower resting heart rate
10. smaller increase in heart rate during moderate work
11. larger stroke-volume
12. more rapid return of heart rate and blood pressure to normal following activity
13. lower blood lactate concentration for the performance of a given amount of work
14. ability to achieve a higher blood lactate concentration before exhaustion occurs

[1]Reprinted from "Physiology of Exercise" by L. E. Morehouse and A. T. Miller, page 278, published by C. V. Mosby Company, St. Louis, Missouri, 1948, with permission of author and publisher.

onary flow will increase commensurately with the increased demand of the heart muscle for oxygen. Until such evidence is forthcoming, we must look for other explanations. If we assume the coronary flow to remain fixed for any given intensity of exercise, and, therefore, also the supply of oxygen, we see the possibility of increasing the work done only if the supply of oxygen is used more efficiently. This appears to be precisely what happens. The clue to this adjustment is found in items 9, 10, and 11 in Table II. We note that, at rest, the heart rate is lower; at a given intensity of exercise, the heart rate is also lower, and the stroke volume is larger, at rest, as well as during the exercise. Now, you will recall that cardiac output is equal to the heart rate per minute multiplied by the stroke volume. It is obvious that in order to get a cardiac output of 10 liters per minute we can have either a heart rate of 200 and a stroke volume of 50 cc., or we can have a heart rate of 100 and a stroke volume of 100 cc. For a given intensity of exercise the untrained and trained individual will have approximately the same cardiac output. The difference will be that the untrained person will achieve his output by a fast heart rate and a small stroke volume, whereas, after training, he will achieve the same output with a lower rate and a larger stroke volume. Is the latter method of achieving the output more economical in terms of O_2 used than the former? Most definitely, yes. The oxygen consumption of the myocardium is nearly linear with the diastolic length of the myocardial fibers, or, to put it another way, with the diastolic volume. Thus, with large diastolic volumes, such as obtain at slower heart rates, the oxygen consumption per stroke is greater, but due to the slower rate the oxygen consumption per unit of time is less. Therefore, with the same cardiac output the slower heart will require less oxygen, or, for the same amount of oxygen used, the slower heart will pump more blood. The work capacity of the heart has been increased, therefore, by making more efficient use of the available supply of oxygen. This appears to be the chief contribution made by training to the work capacity of the heart.

Someone is sure to raise the question as to whether or not cardiac hypertrophy occurs as the result of exercise, and, if so, whether or not this is a beneficial development. It is axiomatic in physiology that use begets hypertrophy and disuse begets atrophy. The heart appears to be no exception to this rule. Hypertrophy, anatomically, consists of a thickening of muscle fibers. Both the amount of sarcoplasm and the number of myofibrils (the contracting elements) increase in each fiber; there is *no* increase in the number of fibers. It is usually made apparent by an increase of the heart weight/body weight ratio. Observations on migratory birds show a greater heart weight/body weight ratio than in nonmigratory fowl; the same is true when comparing working and nonworking breeds of dogs. Experimentally, it has been shown that the heart weight/body weight ratio in dogs can be increased by exercise, and, likewise, in rats. In addition, in the rat it has been shown that regression of the hypertrophy takes place after the rat goes back to a sedentary regime. The data on humans are too few to be certain whether or not the same is true; however, a good case can be made for physiologic hypertrophy.

Whether or not a physiologic hypertrophy is useful is debatable. Fundamentally, a hypertrophy

should be beneficial, because, for the same degree of diastolic fiber length, the contractile force and stroke volume should be greater in the hypertrophied heart. However, we certainly know that the hypertrophy associated with heart disease is a liability rather than an asset. Unfortunately, a greater mass of contracting heart muscle will require a greater supply of oxygen. Whether or not the hypertrophy is beneficial will depend, then on whether there has been a concomitant increase in the coronary circulatory bed and coronary flow. Evidence from pathologically hypertrophied hearts indicates that coronary development does not keep up with the increase of muscle mass; consequently, there is a relative decrease in coronary flow. If this applies to normal hearts hypertrophied as the result of exercise, then it truly represents the limiting factor that determines exercise tolerance.

The Heart as a Factor in a Clinical Exercise Program

Harry D. Bouman, M.D.

The previous paper has given us an excellent review of what exercise will do to the heart, and the question, "What does this mean in a clinical exercise program?" arises. The previous papers have made it clear that, in any exercise program, one will have to raise the question, "How does it load the circulation, and what effect does it have on the heart?"

It is too often the practice when a vigorous exercise program is set up to forget that besides working the muscles that have to be exercised, we are putting a severe extra strain on the circulation, and particularly on the heart, and the previous paper has made very clear what the physiological limitations are in such a program.

Obviously, everyone of the points brought out in the previous paper should be considered in setting up a clinical exercise program, and it has become clear from the preceding discussion that no exercise program can be satisfactorily completed without previous careful evaluation of the status of heart and circulation. From a clinical standpoint there are three main points which I would like to emphasize.

First, it has been pointed out that the factor limiting the heart is the coronary blood flow, and it is very important to realize that there are no physiological means to increase this blood flow. We are all aware of how serious a defect in coronary blood flow can be, and how difficult it is to evaluate clinically its adequacy. Recent measurements by Ivy and Krasnow are an attempt to re-evaluate the adequacy of the coronary blood flow.

Dr. Opdyke pointed out that there is a fixed limit which any normal individual has. Clinically we are all familiar with this, and any normal individual who has ever climbed a steep hill knows the results in pounding of the heart, shortness of breath, etc. If one took the time to feel one's pulse, one would notice probably the most significant phenomena in the increase in heart rate.

It is essential, then, to know the capacity of the heart. This need is accentuated because many of our patients, requiring an exercise program, also have heart disease. Many of them are older individuals with the initial evidences of arteriosclerosis, and others have had heart disease in the form of rheumatic fever or other diseases. It is important, then, to realize that a thorough evaluation of the heart should occur before a strenuous exercise program is started, and also that the response of heart and circulation to the program should be checked frequently as it progresses. Particularly, a frequent check on pulse rate should be made. It is our practice to allow only a limited increase in pulse rate and to watch very carefully for shortness of breath. Electrocardiograms are taken and repeated as indicated during the exercise program.

The second point, which has been emphasized

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in the previous paper and is of considerable clinical importance, concerns the effect of training. It was pointed out that a gradual exercise program increases the function of the heart by increasing the efficiency, decreasing the heart rate, and increasing the stroke volume. Fortunately for us, the adaptation of the heart to an exercise program usually occurs faster than the increased muscle power, so that in many instances the heart has adequate opportunity to keep in step with the increased exercise. However, it should be realized that this will not be so, unless the heart is in a good enough condition to take the load put on it by the exercise program.

Dr. Opdyke pointed out the advantages of different types of exercise in the training of the heart. This brings up the third, very important, point. Namely, that many of the patients whom we start on exercise programs have had a lack of exercise previously. Many of these patients start exercising after long periods of bed rest, due either to the fundamental disease or to a lack of muscle power, such as in neurological diseases, or due to the fact that they have been unable to move around and have, therefore, led a bedridden, or at least resting, life for a long time before they come under our care. It is, therefore, of considerable clinical importance to analyze whether bed rest, in itself, has an effect on cardiovascular function.

Recently, a very interesting experimental investigation of this problem has been published by the laboratory of Dr. Ancel Keys of Minnesota, in which a small number of normal subjects were given bed rest under extremely well-controlled conditions. Six young men were used as subjects. Careful physical examination revealed no noteworthy physical defects. The men were kept in the laboratory during the entire experimental period. During active periods they carried out routine chores connected with the maintenance of the laboratory. They were fed a known diet and were given periods of physical conditioning, three weeks of bed rest, and six weeks of reconditioning.

In the bed-rest period, they were under constant supervision and bedfast nursing care. They were allowed up to ten minutes a day for bowel movements. For an hour in the morning complete inactivity was enforced, and for two hours in the afternoon, and for one hour in the evening. Lights were turned out at a fixed time. The subjects were allowed to read, but a limit was put on letter writing. No "occupational therapy" was allowed. Visitors were limited.

A conditioning program of six weeks was carried out before each period of bed rest. The men walked on a motor-driven treadmill at a fixed speed, at a fixed grade, six days a week. They ran for 3 minutes, at 7 miles an hour, at a 10 percent grade. Daily walks and runs were increased according to a fixed schedule. Psychomotor conditioning was included in the program. Again a rigidly fixed schedule was adhered to.

This program, then, includes a bed rest, and a reconditioning period, under the most rigid laboratory conditions. Needless to say, a rigidly controlled diet was included in the program, adjusted to maintain caloric balance as estimated from body weight. Standard clothing was worn in all tests. It would seem that an experiment on a small number of subjects, controlled so accurately, is considerably more valuable than an experiment on a larger number of subjects without the rigid controls.

A whole battery of tests was performed, and a rigid test sequence was set up for the entire series—postural adjustment, roentgenkymogram of the heart, basal metabolism, aerobic work test, anaerobic work test, psychomotor test, including coordination test, speed of movement tests, tapping tests, reaction time, hand grip, and ataxiameter. Blood volume and cardiac-output estimations were done on separate days. Cardiovascular response to posture was determined by a tilt table. Pulse rates were checked during walking, and, during exhausting physical work, expired air was collected continuously and analyzed. Lactate determinations in the blood were done.

The experiment, then, consisted of a complete

Polio Emergency

Reports of new polio patients in many parts of the country have continued to increase. Local physical therapists have had to assume greater patient loads to meet the emergency.

We are having the most urgent demand for physical therapists for temporary emergency assignments that we have experienced this year, and we have been unable to fill many requests and desperately need physical therapists for emergency polio service.

If you are available, or can be made available, please telephone your chapter president, or **Polio Recruitment Service, American Physical Therapy Association, New York, N. Y., JUdson 6-2932, Collect.**

battery of tests, on a small number of subjects, under the most rigorously controlled conditions of bed rest and reconditioning.

The effect of the prolonged bed rest seems to be most marked in the cardiovascular system. There is a 17 percent decrease in heart volume and an 8 percent decrease in transverse diameter of the heart. There is a highly significant decrease in the resting pulse rate. There was considerable increase in pulse rate, after a half-hour walk, in the patient who had just passed through a period of bed rest. There was no change in mechanical efficiency, during this same walk, as a result of bed rest. The oxygen intake during an effort is reduced 16 percent after three to four weeks of bed rest, and is accompanied by increases in oxygen debt and blood lactate, and a decrease of mechanical efficiency.

Particularly important to us is the realization gained from these experiments that bed rest produces a marked deterioration in the cardiovascular response to posture, as measured by pulse rate and blood-pressure changes produced by tilting. Obviously, this is of particular importance in any attempt toward ambulation of a patient who has had long bedrest. Also, it should be noted from these experiments that bed rest was not severe enough to cause a serious loss of muscle function. In other words, in these experiments we are dealing with patients who, from a muscle standpoint, are still in very excellent condition; yet the heart has already suffered notable deterioration.

It is important then to realize that most of our patients, who come for an exercise program after bed rest, have had considerably longer periods of bed rest than the normal subjects mentioned in this paper. Many of them come to us because of the deterioration of muscle function. Obviously, this becomes of even greater importance in those patients who have had actual heart disease. The setting up of a supervised exercise program for the patient who has suffered acute heart disease then becomes extremely important, not primarily to increase that patient's muscle strength, but primarily to produce gradual exercises to recondition the heart to normal function.

The rate of recovery after the phase of bed rest was roughly proportional to the extent of the deterioration in bed rest. The pulse rate during walking, and the oxygen intake during exhaustive work, recovered first (between two and five weeks). The cardiovascular response to posture was very late in returning and did not usually return until after seven weeks.

In view of the fact that change in posture appeared to be particularly damaging to the heart recovering from bed rest, one would be tempted to conclude that an exercise program after long bed rest, especially in the case of a heart patient, should start with exercises in bed, and that ambulation of the patient should be started relatively late in the program. Old-fashioned muscle-setting exercises are very valuable in this respect. Also, exercises of the arms and hands, while the patient is still lying down, can be used to great advantage. It is obvious that occupational therapy can be given to these patients in the early stages.

The question of when the exercise program, in the case of a heart patient, should start should be decided in consultation between the department of physical medicine and the cardiology department. There will almost always have to be a period of absolute bed rest. The second phase of the exercise program will start with exercises in the horizontal position and exercises to the arms and hands. On the average, this second phase will start about two to three weeks after the acute heart attack, but it is essentially the cardiologist who, on the basis of his heart examination, will have to set the time for the start of this program.

After exercise in bed has been continued for a few weeks, sitting up, and finally ambulation, will be possible. But here again, it is the cardiological examination which should set the time and the rate of increase of the exercise program. We have found in our hospital that an exercise program like this makes the transition from bed rest to ambulation very much easier on the patient, but let me stress again that a program like this, in order to be effective, should be planned through close cooperation between the departments of cardiology and physical medicine.

The physical therapist will have the pulse rate as a continuous indication of what the patient can take during his exercise program, and how much increase in the pulse rate will be allowed should be determined before the program starts. It will be found that, as the program continues, this increase in pulse rate will become less and less.

A third point in relation to the function of the heart in clinical physical medicine, that should be stressed, is the so-called cardiac-neurosis. There are a number of patients, having occasionally had some pain in the left side of the chest and in the left arm, who, with the popular knowledge of medicine which is now widespread, have become extremely afraid that they are suffering from a heart disease. Many of these patients have forced themselves to bed rest and have become invalids because of it. Obviously, patients like these need a very thorough cardiological check-up first, to rule out any evidence of actual heart disease. In some instances, the very fact that the patient has had a good

examination, and has been told by a competent physician that he does not have a heart disease, will make the symptoms disappear overnight. In other instances, the patient has developed such an anxiety-tension state that attention will have to be paid to this secondary involvement. Many of these patients develop spasm of the pectoralis muscle, and in those circumstances, mild diathermy to the pectoralis muscles can be of considerable help.

It should be re-emphasized, however, that this treatment should not be started, under any circumstances, without careful ruling out of any actual heart disease. We have found that, in many of these patients, breathing and relaxation exercises are very useful, provided they are done simultaneously with psychotherapy. Without going into detail about the technic of breathing and relaxation exercises, it might be stressed that relaxation procedures are medical procedures and should be integrated with the entire medical program of the patient, under adequate medical supervision.

Many of these patients with imaginary heart disease have gone through long periods of bed rest and inactivity, and obviously they then fall under the second category and may find it difficult to take up normal life again without a gradual exercise program. We have seen a number of these patients and have found that a gradual exercise program can prove very helpful in treating "heart disease" which was nonexistent in the first place.

The Physiology and Measurement of Peripheral Circulation*

Alrick B. Hertzman, Ph.D.

In any discussion of the circulatory system, it is well to keep in mind that its *chief function* is the *delivery of oxygen*. Circulatory dimensions, design, and operations may be correlated best with this function.

Several basic questions should engage our attention: (1) How well, at any moment, does the circulatory system meet the oxygen requirements of the various parts of the body? (2) How does it meet these requirements? (3) How does failure in the service of this function occur?

It is unfortunate, indeed, that the answer to the first and most basic question can be given only in general terms. We may get some information from the nutritive status of a tissue. It requires little acumen to infer inadequate oxygen delivery in peripheral tissues which ulcerate and die following arterial obstruction. Nor does the sharply reduced work-capacity of ischemic muscle need much elaboration to relate it to the deficit in oxygen delivery. These are obvious phenomena.

Less well understood are the more subtle, long-range effects of a chronic circulatory deficiency. From such may develop, slowly, changes in tissue morphology and function and possibly even in the vessel walls. Such possibilities are indeed legion in number. There is little expansion of such generalities available at the present time.

A valuable approach to this general problem of circulatory adequacy may be made by attempting to measure the *oxygen tension* in tissues. In a sense, this item may indicate whether or not the diffusion of oxygen from the blood vessels is occurring at a required rate. A fall in tissue oxygen tension might suggest that circulatory adjustments could not quite pace tissue needs. This would be valuable information, particularly since one would not need to await the unequivocal, but undesired, answer of the tissues—the development of pathology. It is well to emphasize here that at the pres-

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exit time no test of circulatory adequacy is quite so good, in a scientific sense, as the functional changes which may develop in tissues when suitably severe stresses are presented. But, certainly, neither the physician nor the patient can placidly contemplate such a test. Each desires to be able to predict the condition which may lead to disaster without actually attempting to confirm the predictions.

Some preliminary successes in the study of the *oxygen tensions in tissues* have been won through an adaptation of the oxygen electrode. This gadget consists simply of a platinum electrode mounted in a glass capillary which in turn has been filled with a saline agar gel. If this unit is inserted into an electrolyte solution containing oxygen, and a current passed through the system, oxygen is consumed at the electrode. Diffusion of available oxygen maintains the supply at the electrode. The process therefore becomes dependent on the oxygen tension in the tissues into which the oxygen electrode is plunged. If the oxygen tension in the tissues falls because of impaired circulation, the platinum electrode's consumption of oxygen is impaired and the current diminishes.

THE CAPILLARY PERFUSION

Since the bulk of the nutritive exchange between the blood and tissues takes place across the capillary wall, the measurement of *capillary perfusion* is an objective of major importance in the study of the peripheral circulation. It is important to emphasize that the measurement of the arterial inflow into an organ does not necessarily provide data on the level of capillary perfusion, because of the possibility of the shunting of the capillary bed by vessels which we may designate as arteriovenous anastomosis, or A-V shunts, or thoroughfare vessels. These direct connections (varying in diameter up to 0.1 mm.) between arteries and veins, when open, allow the blood to by-pass the capillary bed in varying amounts. It is therefore possible to consider the flow of blood into any particular part of the body as made up of two fractions: that which by-passes the capillary bed, and may be designated as *shunted flow*, and that which flows through the capillary bed and may properly be called the *capillary perfusion;* very little is known concerning their relative magnitudes in various tissues under different circumstances. Anatomical studies have demonstrated a morphological basis for this problem in several tissues, notably, the skin, pancreas, and kidney.

A first approach to a functional study of capillary perfusion has been made by two types of measurement: the rate of appearance of fluorescein in a vascular bed following its intravenous injection, and, secondly, the rate of "clearance" of Na^{24} from the site of its injection.

The fluorescein method involves injecting this dye into a suitable vein and then recording the progressive increase in its concentration in the tissue whose vascular supply is being observed. The instrumental recording is readily done by illuminating the tissue with ultraviolet light, and following the green fluorescence with a multiplier photo tube whose output can be offered to a suitable recording galvanometer. A time-concentration curve is obtained, the slope of which expresses the rate of escape of the dye into the tissue spaces. If capillary permeability is not altered, and if other factors affecting the procedure are kept well controlled, these curves may express the rate of capillary perfusion.

The rate of uptake of a freely diffusible substance, such as Na Cl, from a perfused tissue should be related to the rate and extent of the perfusion of the capillary bed, providing changes in capillary permeability, or diffusion of the substance along tissue planes, or its drainage via the lymphatics, do not grossly modify the application of this principle. It has been used for years in the saline wheal test introduced by McClure and Aldrich in 1923. This simple but valuable test uses the rate of disappearance of intradermally injected salt solution as a measure of capillary perfusion in the skin. It is still a sound procedure for obtaining information on the cutaneous circulation, although instrumental criteria of the salt uptake would improve its objectivity.

Obviously, this method, in its simple form, is applicable only to the skin. If the rate of uptake of sodium could be followed quantitatively in any tissue, a fairly precise correlation could be established with the level of capillary perfusion. The instrumental methodology used in the detection of radioactive isotopes offers an opportunity to study this correlation. Radioactive sodium (Na^{24}) may be injected into various tissues in extremely low dosage and its concentration at the site of injection followed by a Geiger counter. If one now charts the number of "counts" against time, the slope of the line showing the decrease in the number of counts/minute with time is an expression of the removal of Na^{24}, presumably due to the rate of blood flow.

An important advantage of this technic is its ability to estimate independently the rates of blood flow in superficial and deep tissues. Such discriminatory information is essential, because the regu-

latory mechanisms operating on the cutaneous and muscular circulations may differ radically in timing, direction, and extent of their effects. Likewise, pathological processes and therapeutic manipulations may influence these two vascular beds unequally and in unexpected ways.

These two methods of measuring capillary perfusion offer much promise, particularly when they are combined with simultaneous data on tissue oxygen tension. Possibly the most practical approaches to circulatory problems in the future will be along these or similar pathways. But at the present time, we must be content with less direct methodology.

SOME GENERAL CONSIDERATIONS

When a hydraulic engineer turns his attention to the system of distributing tubes for which he may be responsible, he makes at least two measurements which are essential to his description of the system; he meters the flow in the various tubes, and he also measures the pressures. These data tell him where the fluid is going, what forces are required, and where obstructions or leaks may exist. The study of peripheral vascular states must involve comparable information, if the interpretation of the signs and symptoms is to be on a secure basis, and if therapy is to be selected and given wisely.

The description of a peripheral vascular bed should present data on the following items:

1. The level of *blood flow* and the response of the flow to the various influences which may change the tone of the vessels

2. The *pressure gradient* along the vessels, since this, combined with flow measurements, provides information on

3. The *resistance to flow*. (This item, in turn, indicates whether there is an obstruction to flow, its approximate magnitude, and its location.)

4. The *elasticity of the vessels*. (Aside from its implications respecting the character of the vessel wall, this factor is significant in affecting the form and propagation of the pressure and velocity pulses. These, in turn, influence interpretations of pressure, flow, and resistance data.)

THE MEASUREMENT OF THE PERIPHERAL BLOOD FLOW

Skin temperature as a measure of the peripheral blood flow

The warmth or coldness of the hand or foot is routinely used as a rough guide to the status of its blood supply. Because of the deceptiveness of subjective sensations, many devices have been developed to give increased security to one's impression through instrumental objectivity. Obviously, the expression of skin temperature in °C or °F is more satisfactory than in terms of hot, warm, cool, or cold. We shall see, however, that it does not necessarily follow that cutaneous blood flow is more accurately estimated, simply because surface temperatures have been more closely approximated.

A discussion of this topic should pay attention to: (1) the choice of procedure to be used in the measurement of surface temperatures, (2) the relation of the surface temperature to the peripheral blood flow.

Temperature methodology is a fascinating study in instrumentation. Of the many devices which might be used for our purposes, only two need concern us here—the thermocouple and the radiometer.

The operation of the thermocouple depends on the fact that when two dissimilar metals (e.g., copper and constantan) are joined together, and a temperature difference exists between their "cold" and "hot" junctions, a flow of electrons will continue as long as this temperature difference exists. Further, the electronic diffusion pressure, which we call voltage, is proportional to the temperature difference. Measurement of the voltage generated is simply a question of electrical instrumentation with which we need not be concerned.

The application of these simple facts to the measurement of physiological temperatures is not simple. Thermocouple design, mounting, and application to the skin surface or deeper tissues may modify the thermal environment and conditions of heat loss. The actual temperature of the thermocouple may be as much as a degree or more different from that of the tissue. Hence, concern over the accuracy with which the thermocouple potentials are read may be an instance of unwarranted hair-splitting.

Ideally, the procedure used in the measurement of surface temperatures should not alter them. The radiometer meets this requirement quite well. Its operation depends on the fact that the skin behaves as a black body in the infra-red portion of the spectrum, and that the amount of radiative heat is proportional to the fourth power of the absolute temperature of the radiating surface. Hardy has designed a radiometer which is suitable for clinical measurements of skin temperatures. Radiant heat emitted from the skin surface is reflected along the surfaces of the cone, finally striking the "warm" junctions of a thermopile. The resultant

electric potentials may be then expressed either in cals/cm²/min. or in terms of the temperature of the radiating surface.

It is obvious that at any moment the temperature of the skin surface is determined by the rate of heat loss to the environment, and by the rate at which heat may be gained by the skin, either from the external environment or from the deeper tissues of the body. If heat loss is greater than heat gain, the skin will cool. Conversely, the skin temperature will rise.

In the operation of these principles, the convection of heat to the skin surface by the blood stream is only one of several variables which determine the position of the thermal equilibria at any one moment. Hence, if the surface temperature is to be used as an indicator of blood flow, observational conditions must be defined quite precisely.

Let us first consider the effects of the environmental temperature. If the room air is warmed to 34° C., the skin will be at least that warm, even though no blood flows through it. If blood temperature is 37° C., the maximal rise in skin temperature, with an infinitely large flow of blood, could be only 3°. Further, it can be shown that only a very small flow of blood would be required to increase the skin temperature only 3°. Now, if we lower room temperature to 20° C., we have available a possible increment of 17° in the skin temperature, resulting from increases in blood flow. Although the thermal relations are much more complex in fact than such simple statements suggest, their opportunistic use has led to a conventional environment in which the patient is placed for observation. This environment provides moderately still air at a temperature of about 20° C. Skin of the extremities, without a good blood supply, will cool rapidly under these conditions. If, after cooling, a cutaneous dilatation is induced, a rapid rise in temperature results. The increment in temperature is a rough guide to the extent of the dilatation. But if the experiment is carried out at a summer temperature of 30° C., the increment in temperature is less marked. This does not imply less dilatation.

TABLE I

Regional Skin Temperatures of Nude Subject [at the end of an exposure to a cool environment of three hours (T_a of air = 24° C.)]

Region	T_s	*Region*	T_s
Forehead	32.9	Arm	30.1
Cheek	34.0	Forearm	27.8
Chest	33.1	Finger	25.2
Abdomen	33.7	Calf	30.1
Thigh	32.1	Toe	25.9
Oral	36.2		

TABLE II

Regional Differences in Cutaneous Temperatures and Blood Flows

Region	*Flow cc/cm²/min.*	T_s °C	*Flow* T_c-T_s
Trunk	0.022	34.5	2.3×10^{-8}
Finger pad	0.27	33.6	31.4 "
Toe pad	0.097	27.5	38.8 "

The interpretation of such temperature responses is further complicated by the fact that surface temperatures are not necessarily uniform over the body at any one environmental temperature. When a normal, nude subject is exposed to a cool environment, a surface temperature gradient may be seen along the extremities. *(See Table I.)* The much lower temperatures in the fingers and toes suggest a more intense vasoconstriction there. This phenomenon has been responsible in part for the concept of a *vasoconstrictor gradient* extending peripheral-ward.

These regional differences in skin temperatures necessitate correspondingly different standards in the interpretation of temperature increments resulting from manipulations of the cutaneous circulation.

The indirectness of the skin-temperature criterion of cutaneous blood flow may be illustrated further by comparing estimates of cutaneous blood flow with simultaneous measurements of skin temperature. Table II presents some of our own data as obtained on a normal subject. Blood flows were estimated by a photoelectric technic which is described below. Finger-pad flows were more than ten times higher than in trunk skin; yet finger temperature was slightly lower. Toe-pad temperature was so low as to leave the impression of vasoconstriction; yet blood flow was at a rate four times greater than in the warmer trunk skin. These facts may be re-expressed as the blood flow required to maintain a temperature difference of one degree, between the skin and the environment. The very large regional differences in the value of this quotient indicate that blood flow is only one of several variables which influence skin temperature.

That skin temperature merely indicates the position of the thermal equilibrium between the body and the environment, and tells little or nothing

about how the equilibrium was attained, as illustrated in the common experience that an exercising individual may generate heat at a rate several times above his resting level, with little effect on either oral or skin temperatures. Obviously, he must rid himself of the heat as rapidly as it is generated. The only way available, without a very considerable rise in skin temperature, is through the evaporation of water. He simply must sweat. This would accomplish little, however, without a corresponding increased convection of heat by the blood stream. Skin blood flow should rise. Yet all this can occur with only very moderate changes in skin temperature.

These elementary facts have been known for many years. It occurred to us to attempt their detailed examination in each main region of the body. The identification and quantitation of the several variables which are operating in the setting of skin temperature require the application of the methods of calorimetry.

The heat loss from the surface of the body is made up of the four variables—radiation, convection, conduction, and evaporation. In a simplified experiment, conduction may be eliminated, radiative and convective heat losses estimated from the difference in skin and environmental temperatures, and evaporative rates determined by collecting the water vapor in suitably designed desiccating capsules. Table III exhibits data thus obtained on a nude resting subject in an environmental temperature of 27.6° C. Considerable differences in the rates of heat loss are shown, the highest rate occurring in the cheek. Conservation of body heat by reducing heat losses in the extremities is well illustrated. The regional rates of heat loss may appear to be excessively high, but they are uncorrected for the "effective" surface, which is about 75 percent of the total body.

Full understanding of these thermal data requires an estimation of the cutaneous blood flows. This may be approximated from the fact that, if all of the heat lost from the surface of the skin has been brought there by the blood stream, the blood flow may be computed as follows:

$$\text{the blood flow} = \frac{\text{total heat loss}}{\text{arterial} - \text{venous temperature}}$$

The data in the fourth column of Table III were thus calculated. They indicate high rates of blood flow in head skin and low rates in the calf, which observation would correlate roughly with the differences in skin temperatures exhibited in the corresponding regions.

Before accepting such data on the cutaneous blood supply, it is well to point out certain gaps in information: We do not know, with certainty, what either the rate of cutaneous blood flow or the rate of heat delivery by the blood stream may be. This information requires detailed data on the temperature gradients existing in the tissues. We do not know the actual arteriovenous temperature difference. We do not know at what temperature the arterial blood reaches the superficial vessels, nor at what temperature the venous blood passes into deeper veins.

TABLE III

Regional Rates of Cutaneous Heat Loss and Blood Flow

(Nude subject: T_{air} = 27.6° C. T_{oral} = 37° C.)

Region	*H+C**	*H_{rad}*	*H_{conv}*	*Blood flow cc/M²/min.*
Cheek	64.2	16.2	80.4	900
Chest	49.2	7.2	56.4	280
Forearm	48.0	0.6	48.6	250
Thigh	49.2	6.0	55.2	250
Calf	37.2	7.8	45.0	150

*H = Kg. Cals./M²/hr.

A device for by-passing some of these difficulties is offered in the "thermal circulation index" as introduced by Burton and currently used to express the relations between cutaneous temperature and blood flow. This index, as derived from an analysis of heat flow to the body's surface, is defined as:

$$r = \frac{T_o - T_s}{T_s - T_r}$$

where T_o, T_s, and T_r are oral, skin, and room temperatures respectively. The numerical value of "r" rises sharply at the higher skin temperatures, implying corresponding increases in blood flow.

An additional complication in these thermal and vascular relations is indicated in observations on the Australian aborigine sleeping naked on the ground with air temperatures ranging from 45° to 55° F. Either his heat losses are prevented from rising, or his heat production is increased. Measurements show that the latter does not occur. The only alternative is for this subject to keep heat losses equal to heat production. He may do so by lowering surface temperatures, but there is a practical limit to this procedure because the resulting thermal gradient would extend inward so far as to endanger life. But by reducing heat delivery to the extremities, he may attain the same objective. He is aided in this operation by the fact that the venae comitantes cool the arterial blood. The net

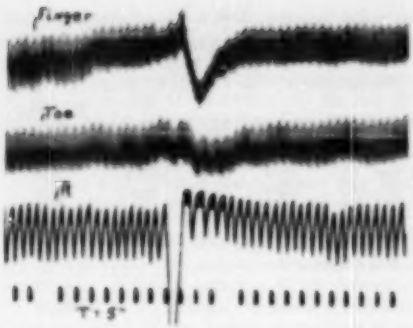

Figure 1. Photoelectric plethysmograms of the finger (upper record) and toe (lower plethysmogram) in a case of epilepsy, after lumbar sympathectomy. Deep breath, as indicated on the respiration record. Time: 5 seconds. (Reprinted from "Applications of Photoelectric Plethysmography to Peripheral Vascular Disease" by A. B. Hertzman and John D. Dillon in American Heart Journal, Volume 20, 1940, page 753, with permission of author and publisher (C. V. Mosby Company, St. Louis, Mo.).)

result is that heat is conserved by transfer to the returning venous blood rather than lost to the outside. The reduction in blood flow may then be less than that indicated by temperature data.

The plethysmograph in the measurement of total extremity blood flow

Total blood flow in a part of an extremity, such as the toe, the foot, or calf, may be measured by enclosing the part in a can, suddenly obstructing the venous return, and measuring the increase in volume which obviously is equal to the rate of arterial inflow. This method is referred to as the venous occlusion method. In practice, it is difficult and cranky; it is hardly a routine technic except in those special laboratories which are suitably equipped with apparatus and technical personnel. The method measures total flow and fails to distinguish, for obvious reasons, between cutaneous and deep muscle flows. This distinction may have considerable practical importance in the interpretation and treatment of such phenomena as rest pains or claudication. Nevertheless, a very considerable fund of information has been obtained by this method.

The photoelectric plethysmograph in the study of the peripheral circulation

The photoelectric plethysmograph is really a photometer with which we may follow changes in the opacity of trans-illuminated tissues resulting from changes in their blood content. Light is supplied by a pencil flashlight bulb. Varying fractions are absorbed in the tissue; only a very small fraction of the incident light is returned to the photo tube. Since tissue absorption of light will remain relatively constant in any one observation unless blood content alters, variations in the latter may be readily followed by galvanometric recording after amplification.

Figure 1 exhibits an example of photoelectric plethysmograms taken simultaneously from the finger and toe. These records illustrate several significant features of plethysmography. Vasoconstriction shows both in decreased blood content and decreased amplitude of the volume pulses. We may expect the latter to parallel blood flow, since these pulses are due to the patency of the small arteries and arterioles, which in turn determine the resistance to blood flow.

One will note the absence of the vasoconstriction in the lower record. This was due to the fact that the subject of the observation had experienced a lumbar sympathectomy some time previous to the experiment. It is obvious that problems in vasomotor, humoral, or pharmacological control of blood vessels may be readily followed by this technic.

The records may be expressed in quantitative terms by a simple procedure. We made the fortuitous observation, early, that the deflections in the photoelectric plethysmogram could be referred to that produced in the record by the insertion of a glass plate in the path of the light. The details of the procedure of quantitating the records are given in previous publications. It suffices here to state that $\Delta V = K_s \cdot V_sH$, where ΔV is the change in blood content of the skin (cc/cm^2), K_s is the volume equivalent of the "filter unit" ($= 2.58 \times 10^{-4}$ cc/cm^2 of skin), and V_sH is the deflection in photoelectric current due to the change in blood content and expressed in "filter units."

Referring to Figure 1, we note again that a vasoconstriction is accompanied by a reduction in the amplitude of the skin pulse. Since constriction also increases resistance to flow, reduction in the latter must also occur. This fact is readily demonstrable with suitable flow meters. We may therefore expect a correlation to exist between flow and the amplitude of the skin pulse. We have found from a considerable number of calibration experiments, carried out in different ways, that this correlation is quite reliable. In any one record, skin blood flow may be calculated after the measurement of

TABLE IV

Topographical Differences in the Basal Rates of Blood Flow in Various Skin Regions

Skin Regions	*cc/cm²/min.*
Group 1:	
Palmar surface	0.052
Plantar surface	0.043
Head	0.12
Group 2:	
Forearm	0.012
Thigh	0.010
Calf	0.013
Trunk	0.012

the skin pulses from the relation: Flow = 0.1 (P), where P is the amplitude of the skin pulse expressed in "filter units." Flow is obtained in cc/cm²/min.

It is instructive to explore the surface of the body with this procedure. Table IV exhibits the resting rates of blood flow in various skin regions. These data may be arranged into two groups as shown, the one exhibiting high rates of flow, the other low rates of flow. We note that the high rate areas are the palmar and plantar surfaces and head skin, and the low rate areas are the rest of the body surface. This division appears to result from the differences in the profuseness of the blood vessels in the respective areas, as may be demonstrated by inducing maximal dilatations in the different skin areas, by suitable dilator influences.

Obviously, the measurement of cutaneous blood flow may be a useful yardstick to apply to various therapeutic procedures, such as the iontophoresis of drugs, diathermy, cold, contrast baths, CO_2 baths, etc. Also, these measurements may have diagnostic value. An illustration of the latter use may be offered here in a patient suffering from Raynaud's phenomenon. Table V exhibits the flow measurements on the palmar surfaces of the first, second, and third phalanges of the corresponding fingers. The flow inequalities were quite striking and correlated well with the complaints of the patient. One will note that the rate of blood flow in the finger pad is only about one-fifth of normal flows at full dilatation, and that the usual ratio of 3:1 for third and first phalanx flows has been reduced. A selective impairment in the arterial supply to the terminal phalanx is implied.

Another illustration is provided in the following case:

A male patient complained of deep muscular pains in the calves of the legs, stiffness in the knee joints, and also pain and stiffness in the thighs. His feet were extremely cold during the winter. Fatigue of the muscles came on quickly in stair-climbing; the calves seemed to tighten or get tense and occasionally become cramped. This symptom picture strongly suggested intermittent claudication with a vascular basis. However, when ischemic work pain was elicited by exercise during occlusion of the leg's blood supply by arm-cuff, the pain disappeared within 5-7 seconds on deflation of the cuff. This is a normal time-interval, one which does not seem compatible with a partial occlusion of blood supply. It seemed desirable, however, to carry out flow measurements. Reflex thermal dilatation increased blood flows in the toes from about 0.1 cc/cm²/min. to about 0.45 cc/cm²/min. This last value is about normal, and, since it was obtained in the toes, was incompatible with main artery obstruction or with obstruction in the minute skin arteries. This conclusion was strengthened by the marked dilatation which could be elicited in the skin of the thigh or calf by dilator drugs. Although one is not warranted in concluding that the muscle blood-supply was normal in this case, another diagnostic approach was indicated.

THE ESTIMATION OF PRESSURE GRADIENTS ALONG THE ARTERIES OF THE EXTREMITIES

It is obvious that a change in the pressure gradient along an artery may be expected wherever the change in resistance to flow occurs. The measurement of the pressure gradients may therefore be utilized as a means of locating the site of such a change in resistance. We have estimated the gradients in systolic and diastolic pressures in the upper and lower extremities by an indirect cuff method which employs an oscillometer with photoelectric amplification of the oscillations. The arrangement which we have employed is extremely convenient, and offers a wide range of sensitivity in recording.

Table VI summarizes some data on pressure gradients in the upper extremity as thus determined. We have indicated the pressure gradients by calculating the ratio of wrist-to-brachial pressures and of digital-to-wrist pressures. It is to be noted that the wrist-to-brachial ratio is the same in normal and hypertensive subjects, and in most of the normotensive subjects with arterial disease. In this particular series of subjects, the presence of arterial pathology had not significantly influenced the propagation of the pressures as far as the

TABLE V

Maximal Blood Flows on Palmar Surfaces of Fingers of Left Hand during Dilatation (Raynaud's syndrome) (Flows: cc/cm²/min.)

	Second Finger	*Fourth Finger*	*Fifth Finger*
First Phalanx	0.052	0.080	0.066
Second Phalanx	0.095	0.037	0.060
Third Phalanx	0.080	0.000(?)	0.137

TABLE VI

Pressure Gradients in the Upper Extremity

	Wrist/Brachial		*Digital/Wrist*	
	S.P.	D.P.	S.P.	D.P.
Normal subjects	1.02	1.09	.86	.88
Normotensive, arterial disease	1.01	1.10	.74	.79
Hypertensive	.98	1.03	.72	.83

radial artery. This is not the case, however, on comparing the ratio of digital-to-wrist pressures. It is to be noted that both the normotensive arterial disease series and the hypertensive series show lower values for these ratios (both for systolic and diastolic pressures) than is true in the normal subject. We also noted that the lowest values for these ratios, as observed on normal subjects, are approximately the average values which were observed in the pathological series, and that very much lower values for the ratios have been obtained in the latter series. These differences in the pressure gradients correlate with differences in the propagation of the pressure pulses, as reflected in changes in the form of the pulse wave as it enters the minute arteries. We are inclined to believe that these data on pressure gradients indicate an abnormally high resistance to flow in the digital arteries of both series of pathological subjects.

Although several methods are available for the designation of the amplitude of the oscillations, as transmitted on to the record from the oscillometer, it has seemed sufficient to designate the amplitude of the oscillations by purely qualitative criteria. Attempts at numerical designation lead to unnecessarily involved procedures whose real advantages are not particularly impressive.

THE COMBINATION OF MEASUREMENTS OF PRESSURE GRADIENTS AND OF CUTANEOUS BLOOD FLOWS

As stated previously, the hydraulic engineer both measures pressures and meters flows in his tube systems. Although a perfect counterpart to his procedure is not available to the physiological engineer, the latter may still obtain useful information by combining some of the methodology referred to above.

Table VII presents some illustrative data from a female patient exhibiting Raynaud's phenomena. One will note that the pressure gradients were approximately normal in the forearm, but somewhat steeper than normal in the hand. It was also noted that the oscillations were considerably reduced at the wrist, more so at the right wrist than at the left, and were very greatly reduced in the fingers of both hands, more so in the right than in the left. These observations indicated obstruction in the main arteries of the hand. The reduction in finger blood flows may be due, of course, either to the resistance in the supply to the main arteries, or to obstruction in minute arteries. Decision between these choices is guided somewhat by additional information in this case. Allen's test for ulnar artery block was positive on the right side. Arteriograms of the right hand of this patient showed a block in the ulnar artery and thin tortuous digital arteries. If the digital artery resistance was solely responsible for the increase in resistance to flow exhibited in this case, one would have expected the brachial/digital pressure gradients to be considerably steeper than the measurements indicated. We therefore believe that small artery resistance was also high in this patient.

Another illustration of the application of these two types of measurements is presented in Table VIII, which offers data on a male patient in which a clinical diagnosis of generalized arteriosclerosis has been made. One will note the small amplitude of the oscillations at the ankle and the foot indicating that there is some obstruction in the main artery at this level. However, the maintenance of the pressures at this level, coupled with the low toe-pad flows at the time of maximal dilatation, suggests that the main resistance to blood flow is located in more peripheral arteries.

Table IX presents relevant data on a male patient with a history of intermittent claudication in both calves. One will note that the pressure gradients are slightly steeper on the right than on the left, and also that the amplitude of the arterial oscillations falls off more sharply on the right than on the left. One will also note that the maximal toe-pad flows are much less on the right side than on the left. As a matter of fact, those on the left are very nearly normal. Palpation of the arteries is in agreement with the implications of the above data. There is, therefore, some evidence of main arterial obstruction on the right side, but the main resistance to blood flow appears to be located in the more peripheral arteries of the right leg. We obtained no evidence of obstruction to blood flow in the left leg, despite the complaint of the patient indicating the existence of obstruction there. There is no disagreement in this with the fact of his complaint, since obstruction in the small arteries supplying the muscles may still exist, despite the negative evidence of these data.

Such a combination of observations on cutaneous blood flows, dilatability of skin blood vessels, pressure gradients, and oscillatory indices in arteries of the extremities permits a description and localization of resistances and obstructions to flow. Our observations, which have been made more or less at random as cases presented opportunity for study, suggest that, in the majority of patients with peripheral arterial disorders, the larger arteries of the extremities are sufficiently involved to impose significant limiting resistances to flow, but that the critical determining resistance, from the viewpoint of tissue nutrition, is usually located in the minute arteries. It has been particularly interesting to note evidence of developing main artery obstruction

TABLE VII

Pressure Gradients and Cutaneous Blood Flows
(Female. Raynaud's syndrome)

Pressure Gradients:

	Wrist/Brachial		*Digital/Brachial*	
	S.P.	D.P.	S.P.	D.P.
Right	1.10	1.15	.67	.64
Left	1.05	1.15	.72	.65

"Oscillatory Index":

Much larger in fingers of left hand and also larger at left wrist than at right.

Finger-pad Blood Flows:

Right middle —0.05 cc/cm²/min.
Left middle —0.19 cc/cm²/min.

TABLE VIII

Pressure Gradients, Arterial Oscillometry, and Cutaneous Blood Flows
(Male. Generalized arteriosclerosis)

	Pressures in Leg	*Arterial Oscillations*
Right knee	140/85	moderately large
Right ankle	120/80	small
Right foot	110/80	small

*Toe-pad Blood Flows:**

RT_1 0.089 cc/cm²/min. } 20% of normal
LT_1 0.088 cc/cm²/min. }

*Measured during reflex thermal dilatation.

TABLE IX

Pressure Gradients, Arterial Oscillometry, Cutaneous Blood Flows
(Male. Intermittent claudication in both calves)

	Right Leg		*Left Leg*	
	B.P.	Oscillation	B.P.	Oscillation
Thigh	130/85	moderate	150/?	large
Calf	128/78	small	150/82	large
Ankle	120/82	small	145/76	large
Foot	110/72	small	135/82	large

Toe-pad Blood Flows:

RT_1 0.085 cc/cm²/min.
LT_1 0.51 cc/cm²/min.

Palpation of Arteries:

	Right	*Left*
Femoral	Palpable	Palpable
Popliteal	"	"
Posterior tibial	Barely palpable	"
Dorsalis pedis	Not palpable	"

even when skin blood flows could be brought to generous levels.

THE PROPAGATION OF THE ARTERIAL PULSE

The essentially static description of flow and pressure data, as cited above, does not provide a complete picture of peripheral circulatory behavior. Due to the intermittent pumping action of the heart, the peripheral resistance, and the elasticity of the blood vessels, arterial blood pressures and blood flows are pulsatile in varying degree. The propagation of these pulses has been studied by many investigators because it was felt that their analysis was essential to a complete hemodynamic description. The majority of these studies have been concerned with the features of the pulse in the larger arteries of the body. In only a few instances have investigators concerned themselves with the propagation of the pulse into the minute ramifications of the arterial tree. Several investigators have utilized photoelectric plethysmographs for this purpose. Matthes, working in Germany, called attention to the fact that the arterial pulse undergoes radical changes in form, on its propagation into the minute skin arteries in instances of arterial disease, and he suggested the possibility that one of the earliest recognizable effects of arterial disease on the propagation of the pulse may be exhibited in this part of the arterial tree.

In our own laboratory we have had many occasions to observe modifications in the propagation of the pulse into the minute skin arteries. The propagation of this wave may be followed by recording its appearance with the photoelectric plethysmograph, which may be applied readily over the radial or digital arteries, and also to the palmar skin. Casual inspection of such records shows little difference in the wave forms of the digital and pad pulses. Also, in the normal subject, digital and radial pulses are almost indistinguishable. On measurement of the records, one notes slight differences in the duration of the upstroke (*i.e.* the crest time) of the radial, digital, and pad pulses. In contrast to these observations on normal subjects, an earlier publication described a tendency of pad pulses to become rounded and plateaued in hypertensive and arteriosclerotic subjects, even when radial pulses were still peaked in form. An extension of these observations to instances of peripheral vascular disease revealed the same phenomenon.

These changes in wave form have been correlated with pulse velocity and other evidence—of resistance to blood flows, and of vessel elasticity.

Space limitations prohibit detailed discussion of such studies.

General Summary

The combination of measurements of peripheral blood flows and of pressures in the peripheral arteries, or their separate use, serves several purposes:

1. It permits an evaluation of the amount of resistance to flow in that particular vascular bed. The resistance is defined in the expression:

$$\text{peripheral resistance} = \frac{\text{peripheral pressure}}{\text{peripheral flow}}$$

If one observes comparable vascular beds (e.g., forearms of adults), the values for resistance can be interpreted in terms of whether blood flows encounter obstructions or not. But they do not necessarily indicate the character of such obstructions, which may be due to spasm of vessels, or organic occlusion, or reduced number and size of the vessels.

2. The dilatability of vessels may be measured. The resultant data evaluate the resistance to flow in terms of the morphological or functional factors contributing to it. Blood vessels may be relaxed in several ways: (a) blockade of vasomotor endings or nerve fibers, (b) decreased tone of the vasomotor supply, (c) *direct* effects of heat, (d) direct effects of dilator drugs. Each procedure serves specific objectives.

Blockade of vasomotor endings, or of synapses at the ganglia, are effected by such drugs as tetra-ethyl-ammonium, priscol, dibenamine, etc. These blockades are often incomplete, and also selective, with respect to the vascular bed involved. Hence, extreme caution is indicated in the interpretation of data obtained with them. Thus, in one of our patients, priscol induced dilatation in the toes which at no time was more than 50 percent of that elicited in reflex thermal dilatation. Frequently, tetra-ethyl-ammonium will induce vasoconstriction in the hand, while dilatation occurs in the foot. Blockade by local anesthetics probably comes close to full dilatation when the entire vasomotor supply is included in the area of injection.

The simplicity of reflex thermal dilatation offers many advantages, but it is not always dependable. Nevertheless, it should be tried early. Surely, positive results would render unnecessary more drastic procedures. Negative results still leave the latter available. We prefer the gentle application of heat, over the entire body, by an electric blanket. Since the objective is to determine whether the skin blood vessels can dilate, it matters not if the part to be observed is also warmed when *flow* measurements are made. When skin temperatures are to be used as criteria of flow, the part must be exposed to cool air. This complicates interpretation, because, in some subjects, local sensitivity to chilling may prevent vascular relaxation.

The study of the dilatability of the vessels by the local application of dilator drugs also offers some interesting advantages. Histamine, mecholyl, pilocarpine, and several other drugs elicit equal and apparently maximal dilatations, when injected into the skin or administered by iontophoresis. The course and extent of the dilatation may be inferred from the hyperemia or measured through estimations of blood flow (thermal circulatory index, photoelectric plethysmograph).

3. The action of vasomotor, humoral, thermal, and mechanical influences on the blood vessels may be studied.

a. *Vasomotor influences.* The outflow and distribution of vasomotor fibers is a key problem in the design of suitable sympathectomies. The selective regional distribution of vasomotor reflexes, and the manner in which they are excited, are determinants of the behavior of the various peripheral vascular beds. Flow and pressure methodology is essential to the investigation of these problems.

b. *Chemical influences.* The actions of vascular drugs, and of sympatholytic and sympathomimetic drugs, may be evaluated in quantitative terms by observing blood flows, simultaneously, in several vascular beds. It is obviously not desirable to dilate a peripheral vascular bed, such as in the foot, at the expense of other more vital areas. Yet, many vascular drugs do this. Neither the autonomic blocking nor the autonomic stimulating drugs act with equal intensity on all parts of the autonomic nervous system. Peripheral circulatory effects may be quite varied in direction, extent, and region affected.

Humoral agents, such as epinephrine and norepinephrine, act unequally on various blood vessels. Sensitization of the effector organ to them, following denervation, poses major problems in sympathetic surgery. Again, flow and pressure methodology provides quantitative observational technics for the study of such phenomena.

c. *Thermal influences.* The direct effects of heat or cold on a particular vascular bed, and the operation of vascular reflexes elicited by the application of heat or cold to some part of the body, pose investigative problems of broad interest. Which vessels dilate or constrict, and how much? what are the relative effects of direct and reflex

thermal influences on these vessels? what are the effects on the distribution of the cardiac output, and on the distribution of the blood volume?—these problems bear directly on widely-used therapeutic methods. The descriptive methods of the hydraulic engineer are essential to their study.

d. *Mechanical influences.* The effects of gravity and its exaggeration in centrifugal force (e.g., in dive bombing); the development of orthostatism as an accompaniment of trauma, disease, surgery, anesthesia, heat, convalescence; the "milking action" of passive and active movements; the effects of intermittent venous occlusion, and of pressure vacuum machines; the effects of stationary position (motor paralysis)—these and many other problems involve peripheral circulatory behavior. Quantitative hydraulic description is needed.

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(Symposium to be completed in the December issue.)

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He is a member of the American Physiological Society, American Heart Association, Society for Experimental Biology and Medicine, and the Inter-American Cardiological Society.

Editorials

It is with a great deal of pleasure that we present in the November and December issues of the REVIEW the complete text of the "Symposium on Physiology" originally given at the Annual Conference in Cleveland. Here, complete in two volumes, you will find papers representing a wide diversity of subject matter, all of it directly related to physiological implications in the field of physical therapy. The list of contributors is a distinguished one, representing a variety of interests, institutions, and activities, composed of names familiar to us all as leaders in the field.

We wish to call particular attention to the introduction by Dr. Harry D. Bouman, Professor of Physical Medicine, University of Wisconsin, who served as coordinator for the symposium. He has given freely of his time and energy, not only in arranging and conducting the original program, but also in collecting and editing the papers for publication in their present form. We wish at this time to express our deep gratitude to him for an excellent job.

We feel sure that this symposium will be received by our members with enthusiasm and pride. The growing emphasis on the importance of physiology in physical therapy has created a demand for and interest in competent, authoritative material dealing with both general and specific problems. All of us have been acutely aware that there have been, and still are, areas in which there is a lack of sufficient, accurate, scientific information relative to physiological principles specifically relating to physical therapy and optimum patient care. It is sincerely hoped that the presentation of these papers will serve as a stimulus to the expansion of interest in varied physiological problems in both laboratory and clinical situations.

In anticipation of the fact that many individuals will want this material in both their personal and professional libraries, arrangements have been made to publish it in book form. Details regarding this will be found elsewhere in the REVIEW.

Dr. Harold Corbusier

It is with deep regret that we report the death of Harold Dunbar Corbusier, M.D., in Santa Fe, New Mexico, on August 31st. Dr. Corbusier was one of the eleven honorary members of our Association, but he was much more than that. "A History of the American Physiotherapy Association" reveals Dr. Corbusier's contribution:

"As early as 1919 there was an incitement to create an organization, and in the latter part of 1920 the movement was gathering force. In September 1920, Dr. Harold D. Corbusier wrote a letter to Mary McMillan suggesting the formation of an organization: 'To advertise to the physician and surgeons of the country the importance of the various methods of treatment by physical means. To elevate and standardize the work and place it on a more substantial basis. . . . To urge upon the various hospitals the importance of establishing departments of physiotherapy. To urge upon the Medical Department of the Army the necessity of creating a reserve force of trained workers and also to endeavor to obtain for the present and former Aides recognition for the work which they have so conscientiously performed.'"

On January 15, 1921, Lt. Col. Corbusier, then of Plainfield, New Jersey, was present at what is considered to be the birth of the American Physical Therapy Association, christened the American Women's Physical Therapeutic Association.

Dr. Corbusier maintained an active interest in physical therapy throughout his lifetime, in spite of the many responsibilities he carried out in relation to his specialty of orthopedics, his connections with the Army, and his international assignments.

Those who attended our annual conference in Boston in 1949 will recall the peivilege of meeting him there. We will never have a more sincere friend.

Have you read the emergency announcement on page 469?

Association Activities

Chapter News

New York: The chapter announces meetings for the coming year as follows: Tuesdays, October 31, December 12, and January 9. Dates for the annual meeting in April and a party in May are to be announced later.

Connecticut: "The Nutmeg News," newsletter of the Connecticut chapter, has announced the following schedule of meetings:

November 21—Hospital for Chronic Illness, Rocky Hill
February 20— Bridgeport
March 20— Newington Veterans Hospital
April 17— New Haven
May 15— Connecticut Society for Crippled Children and Adults, Hartford

New Jersey Chapter Conducts Neuroanatomy Course

The New Jersey chapter of the American Physical Therapy Association is sponsoring an eight-hour course in neuroanatomy during the fall of this year. Lectures will be given by Dr. Charles Nobach, College of Physicians and Surgeons, Columbia University, at the Kessler Institute for Rehabilitation, Pleasant Valley Way, West Orange, New Jersey, on successive Wednesday nights at 8:00 p.m.

Physicians, physical therapists, occupational therapists, and speech therapists are invited to attend. Membership cards to related professional organizations must be presented at each session by all those in attendance. The fee of $15.00 will cover all eight lectures, and single lectures may be attended for $2.00 each.

The following is a list of the lectures and the dates on which each is to be given:

September 27—General plan of the nervous system—central nervous system, peripheral nervous system, autonomic nervous system, the neurone, etc.
October 4— The peripheral nerve—structure, function, degeneration, regeneration. Spinal cord—structure and sensory pathways
October 11— Spinal cord—all pathways, reflexes, upper and lower motor neurone concept
October 18— Pyramidal system—pathway, connections, and function. General discussion of anatomy of brain
October 25— Extrapyramidal system—general aspects of its anatomy, pathways, connections, and functions. Further discussion of the anatomy of brain
November 1— General sensory systems (pain, temperature, touch, etc.)—pathways, connections, and functions
November 8— Special sensory systems (vestibular, auditory, and optic systems)
November 15—General summary—integration of subject matter

Additional information may be obtained from: Miss Genevieve Reilly, Chairman, Education Committee, New Jersey Chapter, 55 Grove Avenue, Verona, New Jersey.

Special Announcement

In this issue of the REVIEW will be found the first part of the "Symposium on Physiology" originally presented at the Annual Conference in Cleveland, Ohio. The remaining papers are to be published in the December issue of the REVIEW which will be available almost immediately following the November issue.

This simultaneous publication plan of the November-December issues has been arranged in order to bring you the entire "Symposium" at one time. This plan will also facilitate a long-contemplated change in the publication schedule of the magazine. In the future, your REVIEW will be put in the mail the 15th of the month *preceding* the date on the cover.

Special plans have been made to have this complete "Symposium" published in its entirety and bound in stiff cardboard for your library. It is expected that there will be a heavy demand for the material. You may secure copies directly from the American Physical Therapy Association in the near future. The date of publication and the price will be announced in the REVIEW.

Membership News

Signe Brunnstrom, Director of Professional Education at the Kessler Institute for Rehabilitation, New Jersey, will leave on October 22nd for Athens, Greece, where for nine months she will conduct a school for physical therapists at the University of Athens. Miss Brunnstrom is being sent to Greece as a Visiting Lecturer by the State Department and the Near East Foundation, under the 1950 Fulbright program. Upon her return, she will resume her duties at Kessler.

From the Intradepartmental News Bulletin, Department of Physical Medicine and Rehabilitation, New York University-Bellevue Medical Center, September 1950: **Barbara White,** who has been assigned to the neurological research project, will assume the position of chief physical therapist at the Institute for Physical Medicine and Rehabilitation on October 1.

Capt. Beatrice Whitcomb. Women's Medical Specialist Corps, U. S. Army, has returned to Brooke Army Medical Center, Fort Sam Houston, Texas, after a year of study in personnel administration at New York University. Captain Whitcomb has been assigned as an instructor in physical therapy at the Medical Field Service School.

Northwestern Graduates Sixteen

Graduation exercises were held for the sixteen members of the 1950 class in physical therapy of Northwestern University Medical School at Thorne Hall on September 29, 1950. Dr. Stafford L. Osborne, Emeritus Professor of Physical Medicine and Chairman of the Department of Physical Medicine, presided. Dr. Leslie B. Arey, Professor of Anatomy and Chairman of the Department of Anatomy at Northwestern University Medical School, gave the address to the graduating class, and Dr. Emil D. W. Hauser, Medical Director of the school, presented the Physical Therapy Certificates to the class.

Each member of the class has transferred from student membership to active membership in the American Physical Therapy Association, and at the close of the program each was presented with an American Physical Therapy Association pin.

The Northwestern University Physical Therapy Alumnae Association gave a dinner in honor of the graduating class at Lewis Towers following the graduation exercises.

New Student Members

The following are newly accepted student members of the American Physical Therapy Association:

Northwestern University

Lois B. Archibald
Mildred Burns
Blanca Elisa Clos
Joanne Crow
Edna J. Foster
Marjorie E. Hoff
Gloria W. LeMaire
Shirley M. Ludwig
Mrs. Margaret Agnes Morley
Margaret McCartney
Jean M. McKain
Margaret McLelland
Marilyn M. Ritz
Eleanor D. Stupka
Barbara Weiss
Judith Welch

Permanente Hospital

Woodson Allen
Esther Anderson
Irvin Barnett
John Beam
Dorothy Clarke
Jean Cummings
John Gressett
Earl Holbrook
Thomas Journey
Morris Mendikoff
David Merry
Marjorie Morden
Bertanne Ogden
Nick Peters
William Santee
Harriet Watts

Graduate Hospital, University of Pennsylvania

Carol Ann Beals
Ralph E. Bell, Jr.
John S. Bishop
Eleanor R. Bloom
Nerreida Cabrera
Robert J. Caleboff
Catherine Copponex
Carter N. Davison
Vilma Evans
Robert S. Foster
Frank J. Fucco
Joseph Galie
Barbara M. Gawthrop
Wilhelmina J. Werkhoven
Edna Elaine Wade
Samuel R. Gehman
Mrs. Ruth M. Hallstead
Joyce K. Hollenbach
Mrs. Laura Lee Greer
Min-Shan Huang
Aleatha R. Jones
Steen L. Merryweather
Eugene Michels
Alice M. Mitsudo
Gloria Peca
William E. Purnell
Louis Riess
Sally W. Sloane
Robert R. Spackman, Jr.
Gordon E. Stermer
Mrs. Biruta E. B. Zimmerman

The death of **Mrs. Mildred Caldwell Nielsen** of Van Nuys, California, on August 12th, has been reported. Mrs. Nielsen had been a member of the American Physical Therapy Association since she completed her training with the Childrens Hospital Society in 1946. She received her B.A. and M.A. in physical education from George Peabody College and had also attended New Mexico State Teachers College and the University of Wisconsin. Mrs. Nielsen had been employed at the Hollywood Presbyterian Hospital in Los Angeles.

School News—Northwestern

From the Northwestern University Physical Therapy Bulletin:

"I think you will all be interested in knowing of the fine representation which Northwestern had at the recent A.P.T.A. Conference in Cleveland. There were 31 NUPT's in attendance. The total A.P.T.A. membership in attendance was 490, which means that over 10 percent of the group were NUPT's. It was remarked that such could be expected since Cleveland is so near to Chicago. However, the NUPT's were there from California and Washington, Florida, New York, and many points between. Also the representation of classes was widespread. Classes of every year except four ('28, '33, '38, '43) were represented. 1940 stood at the top with seven members, and '44 had six members. Even 1950 was represented, with two students attending. This number is also a good representation of the total alumni. From the total of 526 graduates, 16 percent attended the conference. Counting the "casualties" by marriage over the period of 22 years this, too, is a grand showing."

New Brochure Available

The revised edition of "Physical Therapy—A Service and a Career" is now available from the National Office. This new publication, fully illustrated, is entitled "Is This The Door To Your Future?" and contains detailed information concerning training, prerequisite education, and job opportunities. Copies may be secured by writing to the American Physical Therapy Association, 1790 Broadway, New York 19, New York.

New Members

The following are newly accepted members of the American Physical Therapy Association:

William Murray
Nancy A. Bateman
Phyllis Bussero
Alice B. Clay
Mary Lou Goldsmith
Theodore Goodman
Diana G. Griffith
Lois Priscilla Gustafson
Evelyn M. Hallas
Helen Hickey
Mrs. Betty J. Hines
Joseph R. Hyps
Elaine E. J. Jacobs
Samuel Kasser
Mary Katramados
Mrs. Betty M. Lange
Herbert J. Levy
Mrs. Virginia O. Lotheore
Gabriel Wai Chiu Ma
Charles M. Magistro
Theodore Marton
Albert A. McDonald
Rita A. Ouellette
Martha H. Pylka
E. Ruth Reeve
John W. Rhoads
Ruth C. Richardson
Paul Keelan
Jacob S. Schleichkorn
Frances Alice Seyler
Michael W. Sperzano
Medardo Larry Villalobos
John Wasylenko
Claude D. Watts, Jr.
Emily Wellington
Mrs. Miriam Avignon
Michael Phillip Babyak
Janeane Beck
Duane C. Behlen
Helen L. Buch
Patricia J. Busselle
Eileen L. Carter
Mrs. Margaret G. Crist
John Dichkewich
Frank S. Giannattasio
Mary Grimes
James Curtis Gruber
Elinor L. Guza
Mrs. Jane B. Hanrahan
Marie T. Harcharufka
James B. Harrigan
Martha J. Henderson
Rebecca Sue Holton
Keiko K. Iriki
Melvin G. Inspan
Mrs. Geraldine E. Johnston
Maelene Jong
Vernon R. Kisaberth
Atha I. Lighty
Imogene W. Lineburg
Henry S. Low, Jr.
Anne C. Malek
Robert P. Mayberry
Mary F. McDowell
Marie Mikovich
Frederick P. Miller
Mary Mosenthal
Anna M. Mumma
Evelyn F. Musacchia
Lawrence Ray Musgrove
Russell L. Neiswender
Joseph Perez
Grace K. Porter
Mrs. Gunvor H. Queren
Jewel Radford
Clarence W. Rudegeair
Ruth A. Schoenfeldt
Naomi Silverstein
Joseph Starr
Joseph T. Strunk
Elizabeth A. Tachovsky
Lucille Thomas
Dale S. Walker
Marjorie Weinert
Erle L. Williams
Winifred Williams
Joan M. Wilshire
Sally A. Wolleyhan
William A. Winneberger
Martin J. Avignon, Jr.
Louis E. Baker
Miriam Elaine Bent
Mrs. Mazie S. Cawthorne
Roberta R. Donaldson
Harriet A. Dreher
Lois M. Elliot
Dorothy Gorski
Myrtle Gould
Ewing B. Guthrie
Elaine T. Hogan
Janet S. M. Kennedy
Mary H. Meiselwitz
Edward J. Miller
Patricia A. Morrison
Magdalena Ounro
Carmen L. Ortolana
William D. Otterman
Margaret M. Prior
Kathleen Rector
Catherine Rhoades
Mrs. Hazel U. Smith
Alice Torres Balls
Joseph J. Vareka
Robert C. Vickers
Grace P. Walter
Alice E. Williams
Patricia Williams
Rosemary Ann Wiltgen
Lucile Young
Mrs. Winifred M. Zimmerman
Claire Ann Baird
Marta B. Bosch
V. Kenneth Boyum
Leon W. Brouhard
Elizabeth R. Burch
Ruth V. Champe
Sgt. Joseph J. Driscoll
Elizabeth R. Eisenlohr
Daniel Feldhaus
Janet L. Ferguson
D. Rose Funk
Teresa Geisler
Jack M. Hoffroth
Susan Kaufman
Mrs. Sheila J. Kiefer
Manfred E. Lange
Kenneth F. Lonsborg
Alma F. Martin
Angelei Antonia Nunez
Robert J. O'Neill
Blandina V. Phraner
Elizabeth Ann Schultz
Martha J. Schulenberg
Iris J. Sotomayor
Fay Ann Stanton
Barbara E. Teasdale
Frances Wakerman
Marian Wetmore
John L. Gerdes
Alice V. Brodrick
Lena Jenkins
Lt. Pamela Durr
Mrs. Leonora Zukauskas

General News

Deadline for Regular Army Integration

Dietitians, physical therapists, and occupational therapists who formerly served in the Medical Service of the Army are reminded that the deadline date for the submission of applications for integration in the WMSC Regular Army is December 6th, 1950. This opportunity is available to otherwise qualified individuals who are unmarried, who have no dependents under eighteen years of age, and who had not reached the age of thirty-five on day of entrance on duty.

Application blanks and further information may be obtained from The Surgeon General, Department of the Army, Washington 25, D. C. Attention: Chief, Women's Medical Specialist Corps.

Chicago Society Announces Schedule

The Chicago Society of Physical Medicine and Rehabilitation has announced the following schedule of meetings. These programs will begin at 8 p.m., and members of the medical profession are invited.

November 22—Michael Reese Hospital, 29th and Ellis Ave. To be presented by Charles O. Molander, M.D.

January 24— West Side Medical Center (exact place to be announced) Guest Speaker: Arthur H. Steinhaus, Professor of Physiology, George Williams College

February 28— Cook County Hospital, 1835 West Harrison St. To be presented by Disraeli Kobak, M.D.

March 28— West Side Medical Center (exact place to be announced) Guest Speaker: Harold Sofield, M.D., Associate Professor, Bone and Joint Surgery, Northwestern University Medical School

April 25— Hines Veterans Administration Hospital. To be presented by Louis B. Newman, M.D.

May 23— West Side Medical Center (exact place to be announced) Guest Speaker: Frank H. Krusen, M.D., Section on Physical Medicine, Mayo Clinic

National Health Council Announces New Executive

Philip E. Nelbach of New Haven, Connecticut, will assume the post of associate director of the National Health Council, according to an announcement today by Dr. Thomas D. Dublin, executive director. The Council, an association of thirty-one of the leading national health agencies and professional societies, is concerned with the improvement of public health throughout the country and with the coordination of the programs of its member agencies. (The American Physical Therapy Association is an associate member.)

Mr. Nelbach served for four years as assistant professor of public health at Yale University School of Medicine and with the American Public Health Association and the New Haven Housing Authority. He has been consultant to numerous agencies, including local health departments in Connecticut, the New York State Department of Education, the National Housing Authority, and the John B. Pierce Foundation of New York.

In assuming his new position, Mr. Nelbach will make it possible for the National Health Council to augment consultant and advisory services to local and state health councils and to other organizations concerned with community health throughout the country.

Physical Medicine Fellowship Announced

As a part of their extensive educational program, which now has amounted to well over half a million dollars for both undergraduate and graduate scholarships for members, non-members in the United States, and foreign students, Kappa Kappa Gamma, international college women's fraternity, is providing a one-year fellowship for a woman physician from an Occupied Area to advanced study in physical medicine and rehabilitation in the United States. Known as the Harriet Ford Griswold Fellowship, in honor of Mrs. Erwin Griswold of Belmont, Massachusetts, a member of Kappa Kappa Gamma who has a disability resulting from poliomyelitis, the scholarship will provide transportation, food, lodging, and incidental expense for a woman physician, from her home to the United States, for postgraduate study for one year

at the Department of Physical Medicine and Rehabilitation, New York University-Bellevue Medical Center, tuition free. The grant will be administered by the International Society for the Welfare of Cripples.

Selection of the recipient of the award will be made by December 1, 1950, by a committee consisting of Mrs. Griswold; Dr. Howard A. Rusk, Professor and Chairman, Department of Physical Medicine and Rehabilitation, New York University-Bellevue Medical Center; Mr. Donald V. Wilson, Executive Director, International Society for the Welfare of Cripples; and Mr. Eugene J. Taylor, New York City Field Representative, The National Society for Crippled Children and Adults. The necessary arrangements will be made for the recipient to come to the United States during January, 1951, for approximately one year. In addition to the study at the New York University-Bellevue Medical Center, arrangements will be made for the recipient to observe other physical medicine and rehabilitation programs in the United States and to attend professional meetings.

Appointment Information at Army Headquarters

The Chief of the Women's Medical Specialist Corps announces that dietitians, physical therapists, and occupational therapists who desire any information regarding appointment in the Officers' Reserve Corps may now contact Women's Medical Specialist Corps officers assigned in the Office of The Surgeon, Headquarters of the Second, Third, Fourth, and Fifth Army Areas.

Prior to reporting to their new stations, officers so assigned participated in a four-day conference held in the Office of The Surgeon General of the Army. These officers are Lt. Mary Berteling, Office of The Surgeon, Headquarters Second Army, Fort Meade, Maryland; Capt. Christine Ehlers, Office of the Surgeon, Headquarters Third Army, Fort McPherson, Georgia; Capt. Martha Moseman, Office of the Surgeon, Headquarters Fourth Army, Fort Sam Houston, Texas; and Capt. Evelyn Girard, Office of the Surgeon, Headquarters Fifth Army, Chicago, Illinois.

Abstracts

Poliomyelitis and Prophylactic Immunization

Editorial. In THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION, 144:3:240, September 16, 1950.

The subject of anterior poliomyelitis and pertussis and diphtheria immunization has been discussed in many medical circles in comparatively recent months. Its importance is still in dispute.

Epidemiologic case records collected during the course of recent epidemics in the United States have been reviewed for information bearing on this question. To date the data available are inadequate to confirm or refute the British and Australian findings. There is need for further carefully planned studies of the frequency of distribution of paralysis among groups of children immunized, as compared with nonimmunized children in the same community who are as nearly alike as possible with regard to age, susceptibility, and exposure. Furthermore, the question can be approached experimentally. It obviously would be of interest to determine whether intramuscular injections of diphtheria toxoid or pertussis vaccine or both can modify the course of the experimental infection in the monkey.

Postponement of such immunizations for a few months, until the end of the epidemic, would appear particularly discreet when during these few months the children are not likely to be exposed to diphtheria or pertussis, i.e., when these diseases are not unduly prevalent in the community at the same time as the poliomyelitis epidemic.

Common Hand Injuries

F. W. Barden, M.D., Medical Director, Saco-Lowell Shops, Biddeford, Maine. In THE JOURNAL OF THE MAINE MEDICAL ASSOCIATION, 41:7:207, July 1950.

Rehabilitation of a hand injury should begin as soon as the accident has occurred. Proper treatment will help to keep the disability to a minimum. Often, just a chance remark to the injured employee at the time of accident may have a bearing on his future disability and rehabilitation. If splinting is required, only that which is absolutely necessary should be used—keeping the rest of the hand mobile. Passive motion is started as soon as it is safe to do so, and, as soon as possible, hydrotherapy with whirlpool baths is begun. Exercise of the fingers is encouraged by having the patient work with varying-sized rubber balls while receiving the whirlpool. Massage is used considerably. Other exercises also are used to increase finger motion gradually—from gross movement to fine movement. We advocate the use of knitting, crocheting, etc., to facilitate fine finger motion.

Summary

(1) Hand injuries are more prevalent than suspected.

(2) Hand injuries should be given the same "highlighting" in an industrial program as injuries to the eye.

(3) Selective placement of employees will decrease hand accidents.

(4) Proper, immediate treatment of all hand injuries is essential. (Try to give treatment during the "golden period" which is six hours post trauma.)

(5) All wounds to the hand should be considered contaminated at time of injury.

(6) Rehabilitation of hand injuries should begin as soon as the accident has occurred.

(7) The attempt should be made to get the injured employee back on the job as soon as possible.

The Punching Bag in Rehabilitation

J. J. Rudd, M.D., and Reuben J. Margolin, M.A., Boston. In THE JOURNAL OF BONE AND JOINT SURGERY, 32-A:3:698, July 1950.

A valuable piece of exercise equipment for a medical gymnasium—potentially useful, but seldom employed—is the punching bag. It is readily available; the overhead platform drum is easily constructed; and it is inexpensive.

In any exercise program, one of the first factors to be considered is avoidance of the monotony and boredom of the routine prescription. A second factor is the improvement of a specific pathological condition as a result of the activity.

Carefully selected cases of shoulder disability have been treated by the use of the inflated pear-shaped leather bag, in addition to the usual physical, occupational, and corrective therapy. A number of diseases and injuries of the upper extremities, especially in the region of the shoulder and elbow, are amenable to this type of corrective therapy. Patients with subdeltoid or subacromial bursitis, as well as those with dislocation or fracture of the shoulder or elbow joint, can expect gains in strength and in range of motion in specific planes.

In certain conditions, we were able to aid the patient in regaining an appreciable measure of coordination and rhythm. Few gymnasium exercises require a greater synergistic action of muscles than striking the bag. Rhythmic mental responses foster the element of awareness, aid in re-establishing the reflex arc, and combat what may be a very disabling psychological block or "mental alienation."

All punching-bag exercises should progress from the very simple to the more complex. The patient should learn to strike the bag in a manner which will benefit his particular condition.

Rehabilitation of the Amputee

Odon F. von Wersowetz, M.D., and Maude W. Baum. In THE MILITARY SURGEON, 107:1:1, July 1950.

In the total treatment of the amputee, rehabilitation starts before or at the time of the operation and is continued until the patient is returned to the community, prepared to meet satisfactorily the tasks of daily living.

The patient should be prepared psychologically for the operation. This will minimize the mental shock and will prepare him for successful rehabilitation.

Adequate surgery secures a stump which has the optimal length for the proposed prosthesis. Functional rehabilitation in the postoperative period must start at the earliest possible moment, even in spite of an open wound that did not heal by primary union. It is better to commence the use of muscles to move the adjacent joints by the end of the first week, before they have wasted away, rather than to work for months, later, in trying to induce them to regain their strength. The patient should be instructed in good position of the stump, early, to prevent contractures and deformities, which are easily acquired but hard to correct.

The preprosthetic treatment and training prepares the stump for the prosthesis by shrinkage and toughening; the shrinkage is best accomplished by using an elastic bandage and is obtained by planned pressure. This will result in obtaining a properly shaped stump, conical for the standard or conventional prosthesis and cylindrical for the suction socket prosthesis. Muscle imbalance and faulty posture are corrected by active exercises. These exercises should be gradually progressive from mild to heavy resistive.

Proper fitting and alignment of the prosthesis is the most important phase of the rehabilitation. Because shrinkage and seasoning of the stump requires considerable time, it is found from practical experience that the prosthesis should be fitted early. Later the socket should be altered so as to make it conform to changes of the stump. This will prevent faulty habits of walking or function. In addition, early weight-bearing in the lower extremity is essential and is the only practical way to toughen a stump.

Training in the use of the prosthetic device should be under the supervision of a qualified instructor, who has a thorough knowledge of kinesiology and recognition of the possibilities and limitations inherent in the use of a prosthesis. For the lower extremity amputee, the instructor should be a qualified physical therapist; and for the upper extremity amputee, a qualified occupational therapist. During training, especially of the lower extremity amputee, good habits and patterns of motion should be insisted upon at all times. Only under exceptional conditions should substitute motions be permitted. The successful and effective wearing of a prosthesis depends largely on the psychological adjustment of the patient in learning to use it.

Successful rehabilitation of an amputee is not completed and the medical responsibility is not ended, until the patient has obtained maximal training in the activities inherent in daily living and is able to compete economically with normal people, despite his handicap.

Disintegration of Motor Function Resulting from Cerebral Lesions

D. Denny-Brown. In THE JOURNAL OF NERVOUS AND MENTAL DISEASE, 112:1:1, July 1950.

Neurologic disorders indicate that, in so far as disorders of sensory, cerebellar, and cortical structures bring imperfection to willed movement, each of these structures must contribute to its normal performance. Even though destruction of the precentral gyrus in man has a devastating effect on movement of the limbs of the opposite side, the loss is not absolute. Full understanding of the significance of the motor functions of the cerebral cortex awaits more knowledge of the adequate stimulus for activation of each area.

The behavior of the grasp reflex, instinctive grasping, and the placing reaction indicate that the rolandic cortex is concerned especially with the conditioning of proprioceptive stretch-reflexes by contactual effects. The tactile component of this mechanism is not the same as that which perceives touch or pressure, and has tonic, continued effects, as well as special phasic and specific reactions. In the absence of this balance of tactile component in movement, the proprioceptive component is overactive and this constitutes spasticity. Lack of integration between perceived and unconscious sensory factors in movement appears to underlie ideomotor apraxia.

Orienting movements of the head, eyes, mouth, and limbs are served by extrarolandic regions of the cortex. Within this system, removal of the modulation provided by the cerebral cortex, as in frontal lobe lesions, results in some fixity of the righting reactions, giving a general rigidity.

The rolandic cortex also exerts control over the segmental mechanism, but in a sense opposite to that of the extrarolandic cortex, as shown by the opposite primary movement response to stimulation of these two divisions of cortical motor function after pyramid section. Posture and movement are not separate entities, because

all types of movement have the nature of facilitated fragments of postural reactions.

The rolandic and extrarolandic cortex contribute complementary features to movement. Their interrelationship is such that disorder of one leaves unopposed the unwanted activity of the other. Such activity is that which the clinician has habitually called extrapyramidal, but the movements concerned are the result of incoordination between the two systems, and not the special function of one or the other. Breakdown of successively lower levels of integration allows the simpler and more powerful antagonisms, total spontaneous activity of one system versus that of the other, leading to athetosis, dystonia, and parkinsonian tremor, respectively. These motor disorders, like spasticity and rigidity, are each seen as a disequilibrium due to loss of one member of a balanced pair, rather than as the release of a function by loss of an inhibitory suppression of that function. The bizarre postures of athetosis and dystonia have meaning only as conflicting extremes in the pattern of control of the basic organization of the segmental mechanism by rolandic and extrarolandic cortex. There is thus no "center" for spasticity, rigidity, or involuntary movements, nor may a "pathway" be expected for these functions. Each is the outcome of partial or complete release of a normal component in movement from its natural competitor.

Treatment of Thrombophlebitis

Manley B. Cohen, M.D., Legion, Texas. In THE AMERICAN JOURNAL OF SURGERY, 80:1:44, July 1950.

Reports in the recent literature have given an erroneous impression of the meaning of the term "early ambulation" and its relation to thrombophlebitis. Discussions have entailed only the unqualified and restricted sense of the term, usually meaning that the patient is allowed out of bed within seventy-two hours, postoperatively, without necessarily instituting adjuvant measures. Consequently, the confusion which has arisen regarding such therapy requires clarification.

As the problem now is understood, the term, early ambulation, should be discarded. "Early activation" of the patient better portrays what is meant. In this broader sense a definite series of therapeutic measures is included beyond that of getting the patient out of bed. Activation begins while the patient is still in bed and as soon as he has regained function of the extremities postoperatively. A vigorous program of active movements of the toes, feet, and legs and modified quadriceps-setting exercises should be outlined to the patient. Oschner has recommended the use of a board at the foot of the bed against which the patient may exert pressure.

Adams has stated that energetic foot exercises, after proper instruction of the patient and nursing staff, resulted in a 50 percent reduction in thromboembolic disease. Later, in a study of the same groups of patients, Evans and Boller reported that three times as many fatal emboli occurred among patients not exercising as among those who were exercising.

The patient, when he is allowed out of bed, often as early as the day of operation, should be walked. The patient is not allowed to exert himself getting in or out of bed. Dangling should be avoided. Once out of bed he is not permitted to sit in a chair. During the ensuing days the patient is allowed to walk further each day until he is completely ambulant. This is accomplished usually by the fourth or fifth day. To permit a patient out of bed at an early date following operation may be harmful in certain cases. The indications for maintaining patients on continued bed rest are, therefore, particularly important.

The prevention of thrombosis is the important factor in reducing fatalities. Among the prophylactic measures to be used are: (1) early activation, (2) wider use of supportive bandages, (3) avoidance of tobacco pre- and postoperatively, (4) use of anticoagulants whenever indicated, and (5) venous interruption when indicated.

To prevent embolism in established thrombophlebitis, mild heat and elevation of the extremities, together with the use of supportive bandages, are helpful. If a vasospastic factor is present, the use of paravertebral lumbar sympathetic nerve blocks and papaverine are indicated. Depending upon the particular case, anticoagulants or venous interruption are indicated. In a few cases both venous interruption and anticoagulants are recommended.

The Relation between Threshold Voltage and Frequency in Square-Wave Alternating-Current Stimulation

J. W. Duyff and W. G. Walter, Department of Physiology, University of Leiden. In ACTA PHYSIOLOGICA ET PHARMACOLOGICA NEERLANDICA, 1:1:55, January 1950.

The existence of a frequency optimum in AC stimulation of frog nerve, described by Von Kries as early as 1884, could not be explained on the basis of Nernst's theory of excitation. The extension of this theory given by Hill in 1910, however, is in somewhat better agreement with the facts.

In the present paper, an additional argument, based on experiments where the effect of sine wave currents was compared to that of square-wave alternating currents will be brought forward. If the relative inefficacy of a sine wave current of a frequency less than the optimum frequency is indeed due to accommodative changes occurring during the periods of current rise (first and third-quarter periods), we can expect that with square wave currents no frequency optimum will be found, but that, over the entire range, the liminal voltage will increase with frequency. It will be shown in the following that such is indeed the case.

In experiments on nerve-muscle preparations of the frog it was found that, with square-wave alternating currents, the threshold voltage decreases regularly as the frequency is lowered, in contradistinction to the result obtained with sine waves. The experiments furnish an additional argument for the view that the relative inefficacy of low-frequency, sine-wave, stimulating currents is due to accommodative changes during the periods of current rise.

Sterilization of Water in Hydrotherapeutic Tank by Underwater Use of Ultraviolet Rays

Helen Z. Jezn, M.D., and John D. Currence, M.D., F.A.C.P., New York. In ARCHIVES OF PHYSICAL MEDICINE, 31:9:566, September 1950.

The possibility of using underwater ultraviolet radiation for the sterilization of water in hydrotherapeutic practice has been investigated. In order to determine the destructive action of ultraviolet rays, various test microorganisms were used, including E. coli, Staph. aureus, enterococci, Pseudoanthrax bacilli, and T. mentagrophytes.

Under conditions simulating hydrotherapeutic treatment, a large portion of the seeded organisms were destroyed prior to exposure to ultraviolet rays by the action of a temperature of 44 C., by chlorine (0.06 to 0.2 parts per million of tank water) and by the water itself.

In order to obtain a maximum survival of bacteria for an estimation of the true bactericidal power of ultraviolet rays, dechlorinization of water was accomplished by the addition of sodium thiosulfate or broth. Sodium thiosulfate appeared to be preferable to broth for this purpose, because of absorption of the ultraviolet rays by broth which allowed survival of some of the bacteria.

Various numbers of the micro-organisms, up to 2,000,000 per milliliter of water, were subjected to twenty minutes of exposure to ultraviolet rays with complete sterilization.

The underwater use of ultraviolet light can be recommended as a reliable sterilizing procedure in hydrotherapeutic practice. For optimum effects the water should be simultaneously agitated to permit exposure to the rays of every particle of contaminated water.

An Analysis of Paralytic Thumb Deformities

J. Leonard Goldner, M.D., and C. E. Irwin, M.D., Warm Springs, Ga. In THE JOURNAL OF BONE AND JOINT SURGERY, 32-A:3:627, July 1950.

Paralysis of the opposition unit of the thumb is a major disability. The degree of deformity depends primarily upon the anterior-horn cells affected, and the treatment hinges upon the resulting deformity. Furthermore, the status of the thumb is not constant, and the secondary changes which occur in bones, joints, and soft tissues make an ideal end result difficult. The factors of nerve supply, muscle strength, joint contractures, joint stability, and vascular status must all be considered. The interval from onset of disease, the occupation, the general physical condition, and the stability of the remainder of the arm and forearm must be evaluated before reconstruction is attempted. The treatment of each thumb will vary according to the pathological changes present. With these facts in mind, a general classification is outlined, and the variations in treatment are discussed.

Shoulder-Hand Syndrome Following Coronary Heart Disease: Report of Case

I. N. Carroll and M. Mahru. In DELAWARE STATE MEDICAL JOURNAL, 22:43, March 1950. Abstracted in THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION, 143:11:1024, July 15, 1950.

According to Carroll and Mahru, pain in the shoulder region following myocardial infarction is well known, but the shoulder-hand syndrome following infarction is inadequately understood. The mechanism of this syndrome has been summarized by Steinbrocher as being due to widespread disturbances of the internuncial pool, which is composed of a network of widely connecting neurons in the gray matter of the central nervous system, particularly the spinal cord. Stimuli arising in the heart reach spinal-cord segments by way of the cardiac nerves. Here the internuncial pool is activated, and the impulses may travel to involve the anterior horn cells and the posterolateral columns, sympathetic neurons at different levels, resulting in disturbances which cause reflex dystrophy. Either or both of the upper extremities may be involved. The first stage is characterized by swelling, pain, tenderness, increased heat, and discoloration. This may disappear or may progress to the second stage, when the swelling subsides but pain and stiffness become progressively worse. The hands are cold, and the skin becomes thick and attached to the underlying tissues. Muscle atrophy occurs, and trophic changes develop in the bones of the hands and sometimes the shoulder. Clawlike contractures, due to shortening and thickening of flexor tendons, and palmar fascia characterize the third stage. The hands become stiff, hard, cold, and dull rather than shiny as in the earlier stages. The authors describe a man, aged 55, in whom reflex dystrophy developed in both upper extremities. After about ten weeks of physical therapy and elastic traction for the fingers, the patient was able to use his fingers but was not able to perform the finer movements.

Bulbar Poliomyelitis

Maurice Lenarsky, M.D. In ARCHIVES OF PEDIATRICS, 67:6:250, June 1950.

The purpose of this paper is to review the clinical aspects of bulbar poliomyelitis with emphasis on present-day concepts. The importance of this subject lies in the fact that bulbar involvement produces the most serious form of the disease and accounts for almost the entire mortality attributable to poliomyelitis. The spinal type is not lethal, except where there is involvement of the muscles of respiration, and then death may result from either asphyxia, if the respirator is not available, or from pulmonary complications. Bulbar poliomyelitis calls for careful and continuous observation because skillful intervention at the proper time may be lifesaving.

Thus far there are no drugs which will alter the course of the disease. Our experience with darvisul (phenotulfazole) is discussed.

The controversial subject of tracheotomy in bulbar poliomyelitis is discussed.

Transportation of the acute bulbar poliomyelitis patient over long distances may increase the mortality rate.

Effectiveness of treatment of the bulbar poliomyelitis patient requires the cooperation of physicians and workers from allied fields, who form a "poliomyelitis team."

Hyperplastic Arteriosclerosis Versus Atherosclerosis

Eli Moschcowitz, M.D., New York. In JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION, 143:10:861, July 8, 1950.

This paper was stimulated by the serious need of a precise definition of the term "arteriosclerosis" as judged by the diversity of opinions expressed in recent years.

Hyperplastic arteriosclerosis is a process that begins at birth. Evidence has been submitted that the dominant factor in its genesis is the normal intravascular tension and its increased gradient, hypertension. In this concept functional change precedes anatomic change. It is thus impossible to tell when normal aging ends and disease begins. Inasmuch as intravascular pressure cannot be escaped, it is an inevitable destiny of all animals that possess a vascular system such as the human one and who live long enough. It is therefore irreversible, except under most unusual conditions.

This accounts for its universality in all persons, in all climates, in all conditions of sustenance, and in all eras, both ancient and modern. The process may be modified by certain factors, notably the composition of the blood, perivascular stresses and fixations, and the vascular supply of the blood vessels, and in all probability by other contributing factors that remain to be determined. Its universality does not imply that clinical arteriosclerosis and anatomic arteriosclerosis are equivalent. Arteriosclerosis causes disease only when the circulation of a vital organ is impaired. Atherosclerosis cannot be considered as synonymous with arteriosclerosis since it is not primary and lacks the consistent morphologic and/or pathogenetic background of hyperplastic arteriosclerosis. At best it is only a part or a facultative lesion of arteriosclerosis.

The Treatment of Pain and "Spasm" in Poliomyelitis with "Prisoline"

W. O. Geisler, M.D.; W. T. Mustard, M.D.; and C. S. Anglin, M.D., Toronto. In THE CANADIAN MEDICAL ASSOCIATION JOURNAL, 63:1:60, July 1950.

The most damaging effect of poliomyelitis is flaccid paralysis. This paralysis is determined by the extent of anterior horn cell destruction as a result of virus invasion of the central nervous system. There is no available therapy that influences the recovery of the anterior horn cell.

Muscle pain, tenderness, and hypertonic contracture in the acute and early convalescent stages of poliomyelitis is a symptom complex of minor consequence in the absence of paralysis. If paralysis is added, this symptom complex becomes more important as a possible factor in producing deformities. From a study of two groups of children demonstrating pain, muscle tenderness, and hypertonic contracture with added paralysis as a result of poliomyelitis, we do not feel that prisoline demonstrated any particular value as an adjunct to physical therapy.

Rehabilitation of the Lower Extremity Amputee in a Large General Hospital

Ward M. Schultz, M.D., New York City. In NEW YORK STATE JOURNAL OF MEDICINE, 50:17:2061, September 1, 1950.

Rehabilitation of the amputee has been defined as the endeavor to achieve the maximum function and adjustment of the amputee and to prepare him physically, mentally, socially, and vocationally for the fullest life compatible with his abilities and disabilities. It is the objective at Bellevue Hospital not only to fill in the gap between surgeon and limb-maker, but also to carry the patient well beyond this stage, so as to permit that patient the fullest possible life. The physician practicing physical medicine and rehabilitation at Bellevue learns the problems of the surgeon, develops an intimate knowledge of the types and working of available prostheses, studies the problems of the limb-maker, follows through with the training of the amputee on the Rehabilitation Wards, and evaluates his work potentialities.

From the analysis of the data presented, it appears that in a prosthetic program in a general hospital such as Bellevue the following conditions will be met:

(1) At least two-thirds of the amputees seen will be males.

(2) About 70 to 75 percent of the lower extremity amputations will be due to peripheral arteriosclerosis, with or without diabetes mellitus.

(3) About 50 percent will be unilateral, above-knee amputations, and 25 percent unilateral, below-knee amputations.

(4) At least 50 percent cannot adequately use prosthetic appliances because of age, lack of skill, severe intercurrent disease, contractures, or unreliability of the patient.

(5) The ability to perform an adequate swinging-through gait on crutches is a reliable test for the skill necessary to use an above-knee prosthesis.

(6) The degree of skill needed to use a below-knee prosthesis is much less than that required for an above-knee prosthesis.

(7) With the proper case-selection, specific prosthetic prescription to meet the individual need, proper fitting and adjustment, and adequate training, excellent results can be obtained in this type of amputee, even in the older age groups.

Relation of Maximum Grip Strength to Grip Strength Endurance

W. W. Tuttle, C. D. Janney, and C. W. Thompson, Iowa City. In JOURNAL OF APPLIED PHYSIOLOGY, 2:12:663, June 1950.

Strength has been measured by dynamometers of various types since their introduction by Brigham in 1872. In general, strength has been measured either as contraction or breaking strength, expressed as pounds or kilograms. Up to the present time the dynamometers employed provide for measuring maximum strength only. If one wishes to gain a complete picture of strength in man, it seems necessary to record not only maximum strength, but also the tension developed by a group of muscles over a period of time.

A dynamometer for measuring and recording maximum grip strength and strength endurance is described. In order to show the relationship between grip strength and grip strength endurance, data were collected from two hundred university men between the ages of twenty and thirty years. The percentage of the maximum grip strength which was maintained for one minute (strength endurance) was correlated with maximum grip strength. The coefficient for the right hand was —0.40 and for the left, —0.41. This suggests that stronger individuals can maintain a smaller proportion of their maximum strength than those with less initial strength. However, a correlation of maximum strength with the strength endurance index gave a coefficient of 0.67 for the right hand, and 0.66 for the left. This indicates that the individuals with the greater maximum strength have a greater strength endurance index.

The Manipulative Treatment of Low-Back Pain

Howard J. Schaubel, A.B., M.D., Grand Rapids. In THE JOURNAL OF THE MICHIGAN STATE MEDICAL SOCIETY, 49:8:915, August 1950.

The principles of the conservative treatment of low back-ache should include more than rest, heat, braces, and massage. An attempt should be made to understand the pathology in the many cases of low back-ache wherein the X rays and physical examination throw no light on the cause. All joints, whether deeply or superficially situated in the body, are susceptible to similar kinds of trauma, and will react alike to injury as well as to treatment. Low-back pain of indifferent or indeterminate character should be thought of as an intrarticular joint and adjacent soft-parts injury, and treated according to the accepted methods used in the care of extremity joint sprains, strains, and dislocations.

The author has noted no ill effects following the manipulative procedure without anesthesia in over five hundred patients. Spinal manipulation is not presented as a cure for all cases of low-back pain; it is merely another means of attack for a difficult problem, and is not always sufficient in itself.

Spinal manipulation is a useful adjunct in the treatment of low-back pain.

A complete history, review of systems, physical examination, and roentgen evaluation must be done before manipulation is performed.

There are contraindications to manipulation.

Where there is demonstrable pathology other than an intervertebral disc injury or muscle spasm, manipulation is neither performed nor advised.

Patients with poor cardiac reserve constitute a definite risk to manipulation.

Fractures of the Femoral Shaft

Charles Rombold, M.D.; H. O. Anderson, M.D.; H. O. Marsh, M.D.; and J. F. Lance, M.D.; Wichita. In THE JOURNAL OF THE KANSAS MEDICAL SOCIETY, 51:8: 369, August 1950.

The two aspects which make fractures of the femoral shaft an important problem are the economic and treatment factors. The multiplicity of methods of treatment and the total lack of agreement among the advocates of each method, as well as the conflict of reported results of the various methods of treatment, indicate confusion in the strategy of the attack on this problem.

The usual period of recumbency and traction is three months, depending on age, reduction, etc., and largely on some unknown and unpredictable factor of healing of fractures. After adequate callus has been achieved the apparatus is removed and active and passive physical therapy begun. After a week in bed, unsupported, the patient is allowed to walk with crutches, with or without an additional brace support to his thigh, depending on the confidence of the surgeon in the union of the fracture. The period of protection following the removal of the traction usually is about three or four months.

In evaluating his end-results the surgeon may well utilize a formula long in use. The factors of this formula are:

Appearance: visual and X-ray

Function: range of motion in knee, shortening, angulation, subjective complaints

Percentage of economic loss

In conclusion may we emphasize again that in our hands, as well in those of the average surgeon, skeletal traction is at present the method of choice in the treatment of fractures of the shaft of the femur.

Occupational Therapy in Physical Medicine

Barbara Jewett, O.T.R., and M. K. Newman, A.B., M.D., F.A.C.P., Detroit. In THE JOURNAL OF THE MICHIGAN STATE MEDICAL SOCIETY, 49:8:925, August 1950.

Physical therapy, one part of physical medicine, is concerned with the injured part or closely related parts. It administers various forms of mechanical and physical aids in the form of heat, massage, and exercise as stimulation for the therapeutic effect upon the involved part or parts. Progress of the part is recorded. Muscle examination determines the muscle power as a basis of treatment.

Occupational therapy is that part of physical medicine that uses selected activities, physical and mental, definitely prescribed and guided, for the purpose of contributing to and hastening recovery from disease or injury. Occupational therapy contributes to the recovery program by alleviating mental strain. It helps the individual accept his disability or illness and adapt to it. Through acceptable activity the patient can develop or maintain good work habits and thus prevent the establishment of invalid habits. Planned activity aids in the maintenance of physical functions or in the restoration through the redevelopment of joint motion, muscle strength, or coordination. Graded activity improves circulation and encourages the desire to eat and sleep. By improving the posture, and through graded activity, the patient will increase his resistance to fatigue.

In working the patients with physical disabilities, they are instructed and supervised in correct motions to insure a carryover into daily life. The occupational therapist must sense the patients' fears, feel out their interests, analyze their reactions, and adapt the activity and equipment to suit the individual's needs as he progresses. An important part of the rehabilitation program is the training for physical demands of daily life, such as eating, dressing, walking, being able to get about, and writing. Through a testing program the patient's abilities and aptitudes are noted and can be used as helpful information in guiding for vocational rehabilitation.

Rehabilitation of Patients with Hemiplegia

Leonard J. Yamshon, M.D., Los Angeles. In CALIFORNIA MEDICINE, 73:2:181, August 1950.

Patients with hemiplegia can be rehabilitated, unless one or another of the following conditions exists: senility, medical complications prohibiting activity, loss of learning ability, psychosis, lack of motivation, and (sometimes) atonicity.

Motivation is the keystone of rehabilitation. It is of two main types, (1) internal, and (2) external. In internal motivation the impetus to activity arises from within the individual; in external motivation the impetus is provided from the outside. Internal motivation is frequently lost in chronic debilitating illnesses. In order to renew it, external motivation must be supplied as a stimulus.

Rehabilitation is started as soon as medical condition warrants. In cases in which paralysis has been caused by cerebral hemorrhage, activity must wait until all evidence of bleeding ceases.

During the atonic phase, it is important to maintain the range of motion in the joints. Contractures are likely to develop, particularly in older individuals.

Teaching the patient the activities of daily living is started at the onset of treatment. These activities consist of functions which are inherent in everyday existence, such as getting out of bed, standing, dressing, eating, and walking.

Parallel bars may be used in teaching ambulation. The patient first learns to stand and then to walk. Bars are better for this purpose than walkers, since they give the patient a sense of security in that he has something stable to grasp. From the bars the patient is graduated to a cane, and, finally, may be able to walk without support.

Paresis of the arm is more difficult to overcome. Nevertheless, good functional recovery as a result of overcoming atrophy of disuse is not uncommon.

Occupational therapy procedures, which have the advantage of keeping the patient interested in improvement, are best suited to treatment of the arm.

Cerebral Palsy: Medical Considerations and Classification

Temple Fay, M.D., Philadelphia. In THE AMERICAN JOURNAL OF PSYCHIATRY, 107:3:180, September 1950.

Cerebral palsy is a new field of medicine that has emerged in the last five years. It has grown so rapidly that the profession has scarcely had time to recognize it and become fully aware of its progress.

"Cerebral palsy" is a term popularized by Phelps and adopted by the lay public to designate types of paralysis, incoordination, tremor, and crippling effects that arise from lesions of the brain, in contradistinction to injuries or disease of the spinal cord, the peripheral nerves, or the extremities themselves.

The important objectives in this field chiefly concern diagnostic screening and rehabilitation. The lessons learned here, as well as those learned in the management

of acute and chronic phases of cerebral trauma, tumors, and organic lesions of the brain arising in later life, have made possible combinations of physical therapy, pattern movements, and therapeutic exercises that are now known to the profession and have proved to be of benefit to the patient. Adults who suffer a cerebral accident or injury now have the benefit of the work already accomplished in the army rehabilitation centers, and the classification, education, and vocational training so far established for children and young adults.

Diverse as the medical problem may be, greater problems exist in the fields of education and training. Here the physical therapist, the psychometrist, the speech therapist, the occupational therapist, the social worker, and the trained rehabilitationist have attempted to solve the needs for the various ages and for various degrees of handicapping conditions. The educational and rehabilitation groups have been quick to realize the practical difficulties.

The problem is of primary concern to the medical profession. The rapid growth of lay clinics throughout the country has created a great demand for trained medical personnel, trained physical therapists, and speech and occupational therapists. The communities have come to realize that no such trained personnel exists in adequate degree, and no such demand could possibly be met with the means now available for training small groups under personal direction. An attempt has been made to bring to the younger members of the profession the opportunities of postgraduate medical training in this new and extensive field.

Although some of the pioneering work has been done in the field of medicine (Crothers, Phelps, Carlson, and Deaver), there still remains much that must be clarified.

Clinical Electromyography: Physiologic Basis, Instrumentation, Diagnostic Value

Gerald G. Hirschberg, M.D., New York, and Arthur S. Abramson, M.D., White Plains, New York. In ARCHIVES OF PHYSICAL MEDICINE, 31:9:576, September 1950.

Certain aspects of electromyography which are still in the laboratory stage may soon become clinical tools.

Although the importance of electromyography in the study of kinesiology was recognized long ago, little work has been done on this subject. The recording of action potentials gives a sure indication which muscles a disabled patient uses or does not use in certain activities. Such knowledge may be useful for the prescription of corrective exercises and for adequate bracing.

Another aspect to be investigated further is the relation between muscular effort and amplitude of the interfering potential recorded by skin electrodes. There exists a definite relationship. The amplitude of the potential produced by muscle action against maximal resistance may show improvement or further impairment of muscle strength.

A more objective manner of assessing the number of functioning motor units in a partially denervated muscle is electrical stimulation of the motor nerve with supramaximal stimuli. A single diphasic spike is thus obtained, the amplitude of which is determined by the sum of all motor unit potentials in the vicinity of the electrode. This test is of value only in small muscles in which most fibers are within recording reach. Comparison with the same muscle on the opposite side is necessary.

By use of repetitive stimuli at short intervals, fatigability may be determined. Fatigue is shown by a gradual decrease in amplitude of the action potential. Generally the ulnar nerve and the muscles of the hypothenar eminence are used.

Conclusions: (1) Clinical electromyography is of definite diagnostic value in lower motor neuron lesions, in muscular atrophies due to various causes, in localization of spinal cord compression, and in Parkinson's disease.

(2) Clinical electromyography is of definite prognostic value in lower motor neuron lesions.

(3) Clinical electromyography has contributed to the knowledge of the organization of normal voluntary motion and the disorganization of motion occurring in nervous system diseases.

(4) Clinical electromyography is able to contribute to further knowledge in problems of kinesiology, endurance, and power.

Sciatic Scoliosis

Samuel Kleinberg, M.D., New York. In THE AMERICAN JOURNAL OF SURGERY, 80:3:332, September 1950.

In the acute phase, rest and gentle physical therapy are most helpful. These may be supplemented by strapping of the back, wearing of a low-back belt, corset, or a plaster of paris jacket. The affected sciatic nerve should then be stretched by reflexing the limb at the hip with the knee in complete extension. This maneuver should be carried out very slowly. At least ten minutes should be spent in stretching the nerve. This type of manipulation has two dangers, namely, dislocation of the hip and rupture of the sciatic nerve.

When the plaster is removed, the patient is still kept in bed for a week or ten days while the brace is being made. During this time the patient is instructed in mild exercises for his back and leg. These he performs three times a day, increasing the number and strenuousness of the exercises gradually.

When the brace is finished, it is applied and the patient is permitted to sit up in bed or on a chair, then to stand, and finally to walk. The patient's activity is increased slowly, and when he can get about freely, he is discharged to office care. Physical therapy in the form of baking and massage accelerates the recovery. The brace should be worn for three to six months and replaced by a low-back canvas belt. The patient usually can return to his customary occupation, if it is not laborious, in about three months. In those who have to perform hard labor the convalescence should be prolonged at least six months.

Sciatic scoliosis is a symptom complex arising from disorder in the lower back. This clinical syndrome is of frequent occurrence, being found chiefly in otherwise healthy individuals between twenty and fifty years of age. The most important problem in this syndrome is to identify and localize the essential, causal, pathologic lesion. Fortunately, the lesion is readily recognized by a careful history and a methodical examination. The vast majority of patients suffering from sciatic scoliosis can be relieved by conservative measures. Attention has been directed to the value of manipulation and stretching of the back and the affected sciatic nerve in instances of chronic sciatic scoliosis. The technic of the stretching has been described and is recommended because of the high percent of complete recoveries. In the refractory cases of myositis and herniated intervertebral discs, surgery can be utilized to obtain a cure.

Rehabilitation of the Crippled Child

Harriet E. Gillette, M.D., Atlanta. In THE JOURNAL OF THE MEDICAL ASSOCIATION OF GEORGIA, 39:8:332, August 1950.

A crippled child may be defined as an individual in whom there has been interference with the developmental processes, either before, during, or after birth. Such interference may be in the form of developmental defect, trauma, infection, toxins, degenerative process, or other noxious mechanisms, many as yet unclassified. Damage may be in the sensory, motor, or visceral organs and may be so slight as to be merely annoying or so great as to cause total incapacity.

The scope of this paper includes only those children for whom it is good economics to expend a great deal of time, labor, and money in order to make them contributing members of society.

In setting up a program of rehabilitation our ultimate objective is to enable the individual to be self-sustaining and to lead a full life with both vocation and avocation. This should be kept in mind at the first and all subsequent examinations, no matter what the age of the child. The immediate objective is to return the patient to a child's life, consistent with his handicap, as soon as possible.

Poliomyelitis in New York City, 1949

M. Greenberg, M. Siegel, and M. C. Magee. In NEW YORK STATE JOURNAL OF MEDICINE, 50:1057, May 1, 1950. Abstracted in JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION; 143:15:1368, August 12, 1950.

Greenberg and co-workers report an outbreak of poliomyelitis which occurred in New York City in 1949, with a total of 2,446 cases and 179 deaths. About 75 percent of the cases were in children less than fifteen years of age, and 58 percent of the cases were in males. Eleven hundred and nine patients (45.3 percent) showed a spinal type of paralysis, and 392 (16.1 percent) a bulbar or bulbospinal type. The distribution of cases varied in the five boroughs and in the different health districts. The secondary attack rate in homes where a case had occurred was 15 times the attack rate for the city as a whole. Schools opened on schedule, and the ratio of cases in school children to total cases did not increase after the opening. Reports indicated that recent tonsillectomy appears to pre-dispose to the development of poliomyelitis and especially to the bulbar type. Exercise during the onset of poliomyelitis adversely affects its course.

Transfer of Respirator Patients

Clarence W. Dail, M.D., Elizabeth Austin, M.D., O. L. Huddleston, M.D., Ph.D., Albert G. Bower, M.D., F.A.C.P., and Walter Grosse, Los Angeles. In ARCHIVES OF PHYSICAL MEDICINE, 31:9:502, September 1950.

During the 1948 poliomyelitis epidemic at the Los Angeles County Hospital it became necessary to move respirator patients to other hospitals fifteen to twenty miles distant.

Selection of respirator patients to be moved was made by the consulting physiatrist (C.W.D.) in consultation with the chief medical resident. The following considerations were most important: (1) The patient must have been afebrile for at least one week and without complications such as atelectasis, gastric distention, or pulmonary edema; (2) open tracheostomies, Levin tubes, and oxygen administration did not contraindicate movement of the patient; and (3) with proper facilities, patients could be moved who could be out of the respirator only one minute or less.

Success in moving respirator patients resulted only from comprehensive attention to detail so that the patient had every hospital facility present during the time of actual transfer. It also was important to prevent the possibility of mechanical failure, and alternate methods of carrying out all medical procedures were available at all times.

With use of these precautions and procedures eighty patients were moved distances varying from thirteen to fifty miles. All arrived at their destination in good condition and none suffered any delayed ill-effects from the transfer.

Skeletal Suspension in the Treatment of Decubiti

Albert G. Clark, M.D., San Francisco. In CALIFORNIA MEDICINE, 72:6:447, June 1950.

In the usual sacral decubitus, pressure may be relieved by skeletal suspension from Kirschner wires placed in the ilium crests. Pressure sores over the scapulae and thoracic spine may be handled by placing Kirschner wires through the clavicles. Both of these methods lend themselves to the preoperative care of a patient being prepared for the plastic closure of a decubitus.

The Physiologic Analysis of the Psychosomatic Aspect of Peptic Ulcer

Andrew C. Ivy, Ph.D., M.D., D.Sc., LL.D., Chicago. In SOUTHERN MEDICAL JOURNAL, 43:9:814, September 1950.

I have chosen to discuss this particular subject because the aspect of medicine, which is now being termed psychosomatic medicine, had its origin in the field of physiology of the alimentary tract, and we have heard a great deal about this aspect of medicine in the last ten or twelve years. I have read considerable loose experimentation and writing on this particular subject and, for that reason, I wish to present a physiologic analysis of the problem involved.

We must in this particular field insist, as we have insisted in the field of psychoanalysis, that physicians employ the classical logic of science and medicine in drawing conclusions. They are no more warranted to draw conclusions in the field of psychosomatic medicine, on the basis of the *post hoc propter hoc* logic, or on the basis of faulty physiologic and experimental evidence and systematic analysis, than is a person working in any other field.

In considering the subject of psychosomatic medicine we must not be carried off our feet, but apply the same criteria of logic and laws of evidence to this field of medicine that we apply to other fields. Accordingly, at the present time we cannot state unequivocally that any disease is a psychosomatic disease *per se*. According to my analysis of the evidence available at the present time, a tension state is more likely to prove to be an excitatory cause than a basic cause of disease; and if it is ever a basic cause, it operates only on the basis of a constitutional susceptibility of the organ to the disease which affects it.

Ambulation of Patients with Spastic Hemiplegia: Further Observations

Leonard J. Yamshon, M.D., Los Angeles, and Clement C. Thorneycroft, M.D., Van Nuys, Calif. In ARCHIVES OF PHYSICAL MEDICINE, 31:9:599, September 1950.

It has been noted that elevating the sole and heel of the shoe worn on the normal foot of a patient with spastic hemiplegia is a definite aid in ambulation. This alteration of the shoe is not a substitute for the brace. Instead, it is a supplementary aid to the use of the brace. Its effectiveness lies in its inhibitory action on the extensor spasm of the involved leg. This effect is manifested by decreasing the positive support reaction and stretch reflexes, thereby allowing for greater ease in the swing-through of the involved leg and for more adequate use of residual voluntary action. An equally important effect is on the center of gravity. In walking, it is not necessary to shift the center of gravity so far beyond the normal in order to lift the affected leg when the body weight is carried on the unaffected lower extremity. Since weight-bearing on the hemiplegic side is encouraged, the center of gravity is shifted more toward the normal when the body weight is borne on the involved leg. The end result is a smoother gait and greater stability.

In all fifteen cases tested, a definite improvement in gait was seen. The improvement was immediate. Even greater agility in ambulation was accomplished with additional gait training.

The Thimble Electrode: A Device for the Rapid Localization of Motor Points

I. C. Sarnoff and S. J. Sarnoff, M.D., Boston. In ARCHIVES OF PHYSICAL MEDICINE, 31:7:448, July 1950.

The consistency and rapidity of motor point localization was found to be greatly improved by a new device, which provides for the incorporation of a stimulating electrode in a flexible plastic thimble worn on the index finger.

Advances in Research on Poliomyelitis

Herbert A. Wenner, M.D., Kansas City. In ROCKY MOUNTAIN MEDICAL JOURNAL, 47:7:504, July 1950.

There are obvious gaps in information concerning the natural history of poliomyelitis. The methods are crude; a long time is required to establish facts which clearly ring true.

There are several views concerning the dispersion of poliomyelitis virus in a population at risk. Wickman, half a century ago, viewed man as the natural host, and thought that missed cases and healthy carriers were responsible for seeding virus in a population at risk. Another view is that there may be an extra-human source of multiplication of virus, for example, certain species of flies, among others, from which widespread dispersion of virus takes place at certain times. Circumstantial evidence has weighted opinion in favor of the first hypothesis, although it has not been shown conclusively that man is always responsible for the dispersal of virus in the human population.

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The magazine contains 11 articles by leading physicians and physical therapists on research and treatment of polio. There are 16 abstracts of articles on polio from current medical journals.

THE PHYSICAL THERAPY REVIEW—*Special Cerebral Palsy Issue*. May-June 1948 (50c)
This magazine contains 8 articles by leading physicians and physical therapists on research and treatment of cerebral palsy. There are 5 abstracts of articles on cerebral palsy from current medical journals.

PHYSICAL THERAPY REVIEW—*Special Functional Issue*. November 1949 (50c)
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